EMERGENCY ECHOCARDIOGRAPHY

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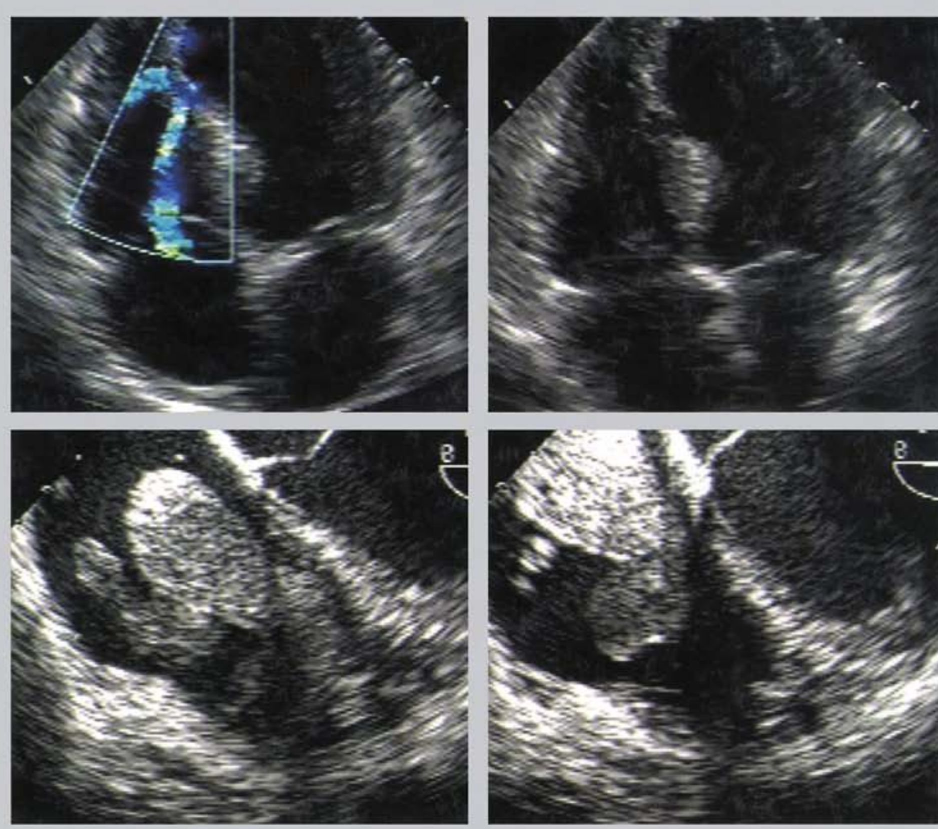

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Emergency Echocardiography

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In memory of Aleksandar D Popović, MD

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Preface

A unique feature of current echocardiography machines is their mobility, which allows examinations to be carried out wherever necessary—from echocardiographic laboratories to emergency departments, wards, catheterization labs, electrophysiology labs and operating theatres. Information about the structure and function of the heart and its hemodynamics can be obtained relatively rapidly and such information is of incomparable value for decision making in cardiovascular emergencies and the critically ill.

This book is intended to outline specific echocardiographic features and procedures pertinent to emergency situations, in particular cardiologic severe acute illnesses. Thus, it is not an introduction into echocardiography in general, and assumes a basic knowledge of the technique. It should serve as a guide to rapid, up-to-date echocardiographic evaluation of life-threatening clinical situations.

Emergency Echocardiography consists of state-of-the-art reviews on the role of cardiac ultrasound in emergency clinical settings. International experts discuss the use of echocardiography in acute coronary syndromes, acute native and prosthetic valve disease, cardiac tamponade, acute diseases of the great vessels, cardiac-related embolism, cardiac arrest, chest trauma and cardiogenic shock. Specific problems related to intraoperative echocardiography, echocardiography in the emergency room and the use of portable echo machines in the emergency setting were also addressed. Since echocardiography is already incorporated into patient management algorithms for the majority of cardiac emergencies, special efforts were made by the contributors to underline its diagnostic power. Importantly, the limitations of echocardiography in specific clinical situations are also discussed. Additionally, the role and potential advantages of special echocardiographic techniques, such as transesophageal echocardiography, are discussed whenever appropriate.

The book is illustrated by carefully selected echocardiographic images and schematics. At times some of the images may appear to be of relatively poor technical quality; however, we included them in the book because they represent images that are obtained under challenging conditions. Some of these are real rarities and contain exciting information.

While we aimed to focus this book toward all members of the health care team, it should be noted that echocardiographic examination in unstable patients in the emergency setting is a highly demanding procedure that requires both excellent technical skills to obtain adequate images in a stressful environment and the ability to interpret findings quickly and accurately. Therefore, despite efforts made to give complete overview of the field to the readers, the importance of appropriate echocardiographic training and personal experience in the evaluation of emergency patients should not be neglected.

Finally, we express our gratitude to all contributors who generously and enthusiastically linked their talent and experience with this project.

The authors sincerely hope that this book will be found useful in everyday clinical practice and encourage feedback, criticism and suggestions.

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1
Echocardiography in acute aortic insufficiency

Edmund A Bermudez and Michael H Picard

Key points

- Acute severe aortic insufficiency (AI) is an uncommon yet life-threatening condition that necessitates prompt recognition and management.
- Echocardiography has become an indispensable tool to confirm the presence of aortic insufficiency, assess its severity, and provide information regarding the etiology.
- In contrast to chronic decompensated AI, in which the left ventricle (LV) is dilated, and the left ventricular ejection fraction (LVEF) is reduced, in acute aortic regurgitation (AR) the LV size may be normal and LVEF is hyperdynamic.
- Acute AI can be the result of various abnormalities of the aortic valve and/or the aortic root, including proximal aortic dissection (type I or type A), endocarditis of the aortic valve, trauma, or aortic prosthetic valve dysfunction.

Acute severe aortic insufficiency (AI) is an uncommon yet life-threatening condition that necessitates prompt recognition and management.[1] Fortunately, echocardiography is a widely available noninvasive modality that affords prompt diagnosis and invaluable information for management of these often critically ill patients. The two-dimensional and color Doppler evaluations remain essential components of the echocardiographic examination in these patients. As such, echocardiography is an important tool in the diagnosis, assessment, and management of patients presenting with acute AI of any etiology.

Pathophysiologic mechanisms

The clinical presentation of acute AI is often dramatic and relates to a sudden volume load placed upon an unsuspecting left ventricle (LV). Because of this, left ventricular end-diastolic pressure (LVEDP) is suddenly and often markedly increased.[2] The LV now operates on the much steeper portion of a normal end-diastolic pressure-volume relationship curve (Figure 1.1). Therefore, the increase in left ventricular end-diastolic volume (LVEDV) results in a much higher end-diastolic pressure. As a result, left atrial pressures may become markedly elevated with subsequent pulmonary congestion. These changes are exaggerated in patients with a noncompliant LV at baseline (such as hypertrophy from hypertension or aortic

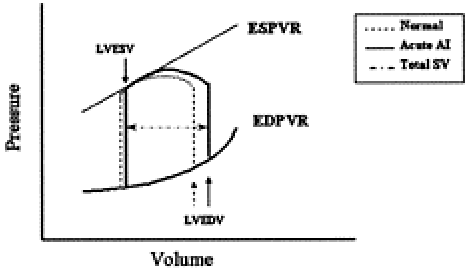


Figure 1.1 Left ventricular pressure-volume loop in acute aortic insufficiency. ESPVR, end-systolic pressure volume relationship; EDPVR, end-diastolic pressure volume relationship; LVESV, left ventricular end-systolic volume; LVEDV, left ventricular end-diastolic volume; SV, stroke volume.

stenosis), as the LV already operates on a steeper end-diastolic pressure volume relationship curve.[3] As such, acute insufficiency in these individuals frequently is more marked and results in rapid hemodynamic collapse. In extreme cases, LVEDP equilibrates with aortic diastolic pressure. In contrast, the LV end-systolic pressure and systemic systolic pressure usually exhibit little change.

Compensatory dilation of the LV is limited in acute insufficiency, and usually results in a near normal-sized left ventricle.[4] However, LV end-diastolic volume does increase, depending on the severity of the regurgitation, and results in an increase in total LV stroke volume (SV). Despite this, the effective or forward stroke volume is reduced, as the regurgitant volume returns to the LV. This is in contradistinction to chronic decompensated AI, where the LV is dilated, the LVEDV is markedly increased, and the total and forward stroke volumes are reduced with a reduction in left ventricular ejection fraction (LVEF).[5]

Clinically, patients are dyspneic, tachycardic, with peripheral vasoconstriction. There is a paucity of the physical findings that are commonly found in chronic regurgitation. The pulse pressure is usually normal with a short aortic diastolic murmur occurring only in early diastole. The first heart sound (S1) is usually soft in severe insufficiency, as there is early diastolic closure of the mitral valve from the regurgitant aortic jet. Occasionally, mid-diastolic closure of the mitral valve may be heard. An Austin Flint murmur and an aortic outflow murmur may also be found.

Etiology

Acute AI can be the result of various abnormalities of the aortic valve and/or the aortic root. Most commonly, acute insufficiency can be the result of proximal aortic dissection (type I or type A), endocarditis or trauma of the aortic valve, or aortic prosthetic valve dysfunction. When acute AI is the result of aortic dissection, proximal aortic dissection may produce valvular insufficiency via incomplete leaflet closure due to aortic root dilation, leaflet prolapse, or a dissection flap prolapsing through normal leaflets inhibiting leaflet closure.[6] Endocarditis involving the aortic valve can produce acute AI when leaflet destruction produces leaflet perforation or when normal leaflet coaptation is prevented. Trauma to the aortic valve may result from nonpenetrating chest trauma, or it may rarely occur during cardiac catheterization.[7,8] Spontaneous aortic valve rupture has been reported to be related to fenestrations of the aortic valve; however, this is very uncommon.[9]

Prosthetic aortic valve dysfunction can result in acute AI in mechanical valves when acute thrombus formation or chronic pannus overgrowth results in an inability of the prosthesis to close properly. Acute AI can occur in bioprosthetic aortic valves as a result of endocarditis (as described above for native valves) or an acute leaflet tear. The normal degeneration of bioprosthetic aortic valve leaflets more typically results in a mild degree of AI which progressively increases. However, if this is unrecognized and not treated, it can progress to severe AI as in the presentation described above.

Echocardiographic evaluation

Echocardiography has become an indispensable tool to confirm the presence of AI, assess its severity, and provide information regarding the etiology. The presence of AI can easily be ascertained from color and spectral Doppler echocardiography. Similarly, the severity of AI in the acute setting can be determined by a combination of two-dimensional and Doppler echocardiography (Table 1.1).

The color Doppler examination of the aortic valve provides the simplest approach for the determination of

Table 1.1 Echocardiographic signs in hemodynamically severe acute aortic insufficiency

Color Doppler measurements
Parasternal long axis view
Proximal color jet height to left ventricular outflow tract (LVOT) height ratio of >0.6
Late diastolic mitral regurgitation
Parasternal short axis view
Color jet to LVOT area ratio of >0.6

Color jet area to LVOT area ratio of >0.6

Regurgitant orifice area (ROA)≥0.30 cm^2 by PISA

Spectral Doppler measurements

Continuous wave Doppler

Aortic valve
Dense regurgitant velocity envelope compared to forward flow velocity envelope
Pressure half-time of regurgitant envelope of <250 ms
Regurgitant volume≥60 ml
Regurgitant fraction≥55%

Descending aorta

Holodiastolic flow reversal

Mitral valve

Restrictive mitral flow pattern

Two-dimensional and M-mode measurements

Parasternal long axis view

Early presystolic closure of the mitral valve

the presence and severity of insufficiency.[10] The characteristics of the color flow jet in the LV outflow tract provide information about the vena contracta. The vena contracta, the narrowest width of the jet as it exits the regurgitant orifice, provides an indirect yet accurate measure of the regurgitant aortic orifice and is relatively independent of flow. For AI assessment by transthoracic echo, this is best visualized from the parasternal long axis view. In the parasternal views, the ratio of the height (long axis) or area (short axis) of the regurgitant jet to the left ventricular outflow tract (LVOT) measurements is thus used to determine severity. If either ratio is greater than 0.6, severe insufficiency is considered to be present.[11,12] The proximal isovelocity surface area (PISA) method[13,14] may also be used to calculate the regurgitant orifice area (ROA) by the following formula:

$$\text{ROA} = \frac{\text{Flow rate}}{\text{Peak AI velocity}}$$

where flow rate=2π(r2)×aliasing velocity and r= aliasing radius. By this formula, an ROA of >0.3 cm^2 may be considered severe regurgitation.[13]

Two-dimensional and spectral Doppler tracings are important for the determination of severity of regurgitation. The density of the envelope and short pressure half-time (<250 ms) of the aortic signal usually signify hemodynamically severe insufficiency.[15,16] The short pressure half-time of the AI velocity profile on continuous wave Doppler is not a specific finding of severe AI, since it is determined by not only the amount of regurgitation but also the compliance of the receiving chamber (LV) and the peripheral aortic resistance. Thus, a mild degree of AI in a stiff LV can result in a shortened pressure half-time. Holodiastolic flow reversal in the descending thoracic aorta will also

be seen in severe insufficiency. Regurgitant fractions and volumes may also be calculated to determine severity by a variety of methods.

Echocardiographic assessment of the mitral valve will also provide clues about hemodynamically significant aortic regurgitation. The transmitral flow patterns from continuous wave Doppler in the apical views may show a restrictive pattern, signifying early rapid equilibration of left atrial and LV diastolic pressures.[17] In acute AI, early mitral valve closure may occur in mid to late diastole as LV diastolic pressure exceeds left atrial pressure.[18,19] If this occurs, diastolic mitral regurgitation may be seen, and further signify acute severe insufficiency of the aortic valve.[20]

Many of these signs of severe AI may be seen in chronic severe AI. However, restrictive transmitral flow patterns, early mitral valve closure, and diastolic mitral regurgitation may be more specific to acute AI. Importantly, the size of the LV is essential to note, as the size of the LV in acute AI is often near normal, in contradistinction to chronic severe AI, where the ventricle is markedly dilated.[21] However, ventricular dilation may be seen in acute AI in the presence of previous valve or coronary disease that may have contributed to LV dilation.

These parameters mentioned can often be elucidated by transthoracic echocardiography (TTE). However, the clinical setting may dictate the use of transesophageal echocardiography (TEE) for the determination of etiologic mechanisms of insufficiency. In many centers, TEE is the modality of choice in the evaluation of aortic dissection, and it can determine the presence of dissection, type, location of entry and/or exit flow of the false lumen, and the severity and mechanism of AI. Furthermore, TEE may be a more sensitive modality for the diagnosis of valvular endocarditis and its complications, and may be chosen directly, in certain clinical settings, without initial transthoracic evaluation. TEE is invaluable in the assessment of prosthetic valve AI in order to understand the mechanism and severity. Therefore, the use of TTE or TEE in the evaluation of acute AI will depend on the clinical setting, operator experience, availability, and regional practice patterns.

TEE evaluation of proximal aortic dissection accompanied by AI provides important information regarding the etiologic mechanisms of insufficiency. In this setting, distinct mechanisms for insufficiency may be present. For example, dilation of the sinotubular junction may result in incomplete leaflet closure secondary to leaflet tethering and a persistent diastolic orifice. Alternatively, leaflet prolapse will produce AI when the dissection extends into the root and disrupts normal leaflet coaptation. This frequently results in an eccentric regurgitant jet. Finally, if a dissection flap prolapses across normal aortic leaflets, insufficiency will result (Figures 1.2 and 1.3).

Echocardiography as a guide to management

Elucidation of the mechanism of acute AI, especially in acute dissection, is important, as this may influence surgical approach.[6] When there is geometric distortion of the valve, as in aortic root dilation, repair with valve preservation may be feasible. In contrast, when fixed abnormalities are found by TEE, replacement is usually recommended. While prompt surgery for proximal aortic dissection is well established, the approach (that is, replacement versus repair) to the associated AI is less clear. However, when reversible

mechanisms for insufficiency are made apparent by TEE, repair may be a feasible surgical approach. In this manner, several options may exist: (1) when incomplete leaflet closure is seen to be due to a dilated root, valve

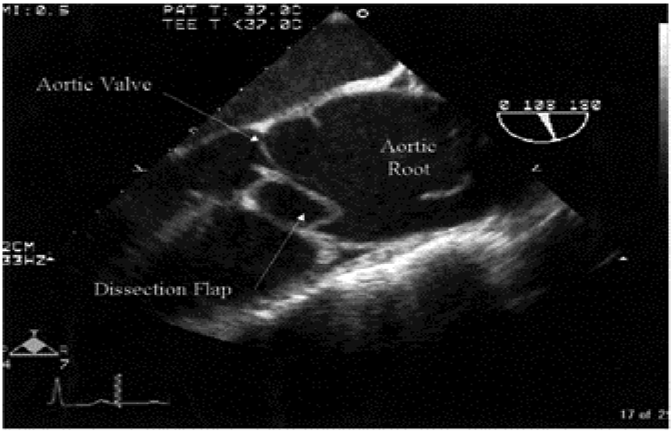


Figure 1.2 Transesophageal echocardiographic image of the long axis of the ascending aorta demonstrating a type I acute aortic dissection with the dissection flap prolapsing through the aortic valve. This results in significant aortic insufficiency due to an abnormality of valve leaflet coaptation.

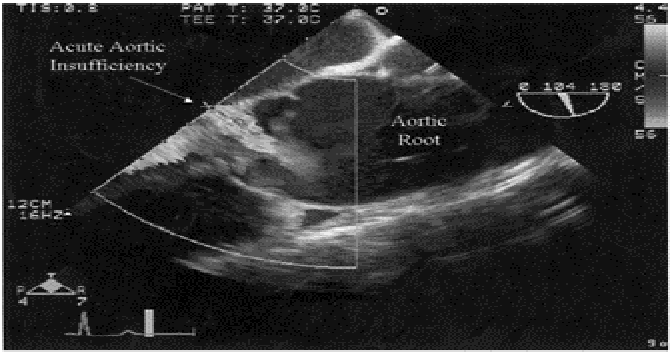


Figure 1.3 Color Doppler illustration of moderate to severe aortic insufficiency on transesophageal

echocardiography of the patient
described in Figure 1.2.

repair may involve narrowing of the dilated aortic root with a graft;[22] (2) resuspension of aortic leaflets at the commissures can be performed when leaflet prolapse is seen; (3) when intimal flap prolapse is seen to cause AI, corrective repair may involve replacement of the ascending aorta without aortic valve replacement.

If endocarditis is complicated by severe acute AI, surgery in general should not be delayed when hemodynamic compromise is clinically evident.[23] Perioperative TEE can elucidate the mechanism of AI, which frequently results from valve destruction and leaflet perforation. Aortic valve replacement is the standard in this setting.

Summary

Acute severe AI is a medical and surgical emergency that often complicates acute aortic disease or aortic valvular endocarditis. Echocardiography provides a convenient and simple method to determine the diagnosis and severity of insufficiency, as well as the etiologic mechanisms involved. This information is invaluable in surgical decision making when valve preservation is preferred in the setting of aortic dissection.

References

1. Rahimtoola SH. Recognition and management of acute aortic regurgitation. Heart Dis Stroke 1993; 2:217–21.
2. Carabello BA, Crawford FA Jr. Valvular heart disease. N Engl J Med 1997; 337:32–41.
3. ACC/AHA guidelines for the management of patients with valvular heart disease. A report of the American College of Cardiology/American Heart Association. Task Force on Practice Guidelines (Committee on Management of Patients with Valvular Heart Disease). J Am Coll Cardiol 1998; 32:1486–588.
4. Morganroth J, Perloff JK, Zeldis SM, Dunkman WB. Acute severe aortic regurgitation. Pathophysiology, clinical recognition, and management. Ann Intern Med 1977; 87:223–32.
5. Braunwald E. Heart Disease: A Textbook of Cardiovascular Medicine, 6th edn. Philadelphia: WB Saunders, 2001.
6. Movsowitz HD, Levine RA, Hilgenberg AD, Isselbacher EM. Transesophageal echocardiographic description of the mechanisms of aortic regurgitation in acute type A aortic dissection: implications for aortic valve repair. J Am Coll Cardiol 2000; 36:884–90.
7. Javeed N, Shaikh J, Patel M, Rezai F, Wong P. Catheter-induced acute aortic insufficiency with hemodynamic collapse during PTCA: an unreported complication. Cathet Cardiovasc Diagn 1997; 42:305–7.
8. Unal M, Demirsoy E, Gogus A, Arbatli H, Hamzaoglu A, Sonmez B. Acute aortic valve regurgitation secondary to blunt chest trauma. Tex Heart Inst J 2001; 28:312–14.
9. Akiyama K, Ohsawa S, Hirota J, Takiguchi M. Massive aortic regurgitation by spontaneous rupture of a fibrous strand in a fenestrated aortic valve. J Heart Valve Dis 1998; 7:521–3.
10. Ekery DL, Davidoff R. Aortic regurgitation: quantitative methods by echocardiography. Echocardiography 2000; 17:293–302.

11. Perry GJ, Helmcke F, Nanda NC, Byard C, Soto B. Evaluation of aortic insufficiency by Doppler color flow mapping. J Am Coll Cardiol 1987; 9:952–9.
12. Tribouilloy CM, Enriquez-Sarano M, Bailey KR, Seward JB, Tajik AJ. Assessment of severity of aortic regurgitation using the width of the vena contracta: a clinical color Doppler imaging study. Circulation 2000; 102:558–64.
13. Tribouilloy CM, Enriquez-Sarano M, Fett SL, Bailey KR, Seward JB, Tajik AJ. Application of the proximal flow convergence method to calculate the effective regurgitant orifice area in aortic regurgitation. J Am Coll Cardiol 1998; 32:1032–9.
14. Thomas JD. Doppler echocardiographic assessment of valvar regurgitation. Heart 2002; 88:651–7.
15. Teague SM, Heinsimer JA, Anderson JL, et al. Quantification of aortic regurgitation utilizing continuous wave Doppler ultrasound. J Am Coll Cardiol 1986; 8:592–9.
16. Labovitz AJ, Ferrara RP, Kern MJ, Bryg RJ, Mrosek DG, Williams GA. Quantitative evaluation of aortic insufficiency by continuous wave Doppler echocardiography. J Am Coll Cardiol 1986; 8:1341–7.
17. Vilacosta I, San Roman JA, Castillo JA, et al. Retrograde atrial kick in acute aortic regurgitation. Study of mitral and pulmonary venous flow velocities by transthoracic and transesophageal echocardiography. Clin Cardiol 1997; 20:35–40.
18. Botvinick EH, Schiller NB, Wickramasekaran R, Klausner SC, Gertz E. Echocardiographic demonstration of early mitral valve closure in severe aortic insufficiency. Its clinical implications. Circulation 1975; 51:836–47.
19. Meyer T, Sareli P, Pocock WA, Dean H, Epstein M, Barlow J. Echocardiographic and hemodynamic correlates of diastolic closure of mitral valve and diastolic opening of aortic valve in severe aortic regurgitation. Am J Cardiol 1987; 59:1144–8.
20. Downes TR, Nomeir AM, Hackshaw BT, Kellam LJ, Watts LE, Little WC. Diastolic mitral regurgitation in acute but not chronic aortic regurgitation: implications regarding the mechanism of mitral closure. Am Heart J 1989; 117:1106–12.
21. Mann T, McLaurin L, Grossman W, Craige E. Assessing the hemodynamic severity of acute aortic regurgitation due to infective endocarditis. N Engl J Med 1975; 293:108–13.
22. David TE, Feindel CM, Bos J. Repair of the aortic valve in patients with aortic insufficiency and aortic root aneurysm. J Thorac Cardiovasc Surg 1995; 109:345–51.
23. Sareli P, Klein HO, Schamroth CL, et al. Contribution of echocardiography and immediate surgery to the management of severe aortic regurgitation from active infective endocarditis. Am J Cardiol 1986; 57:413–18.

2
Echocardiography in cardiac tamponade

Mauro Pepi, Gloria Tamborini and Manuela Muratori

Key points

- ZEchocardiography allows bedside detection, semiquantitation and localization of pericardial effusions.
- Cardiac tamponade is a clinical diagnosis, and may occur with pericardial effusions of various size.
- Echocardiography may reveal warning signs that suggest tamponade physiology, including right ventricular (RV) and right atrial (RA) collapse, inferior vena cava plethora, and increased transmitral and aortic flow reduction during inspirium.
- The duration of collapses is directly related to the severity of tamponade.
- Right-sided collapse may be insensitive in right ventricular hypertrophy and high RV intracavitary pressures.
- Localized pericardial effusion or clot may occur after cardiac surgery. These effusions may be difficult to explore, and modified, off-axis transthoracic echocardiographic views should be used. In poor or inconclusive images, transesophageal echocardiography should always be performed.
- Percutaneous pericardiocentesis guided by echocardiography is an effective and safe procedure.

Cardiac tamponade is a life-threatening condition which requires urgent therapeutic intervention. This chapter covers the role of echocardiography in the detection of pericardial effusion and signs of cardiac tamponade as well as the contribution of ultrasound technique in the management of the syndrome.

Definition and physiology

Cardiac tamponade is defined as significant compression of the heart by accumulating pericardial contents (effusion fluids, clots, pus, and gas, singly or in combination). When liquid is injected into the pericardial sac of an experimental animal, intrapericardial and right and left atrial pressures begin to rise equally; this may occur also in the presence of a small amount of fluid (20–40 ml). As more fluid is added, cardiac stroke volume falls while cardiac output falls less because of compensating tachycardia. In fact, the output of both ventricles depends on adequate diastolic filling; normally, intrapericardial pressure is zero or slightly negative, and the transmural pressure gradient across the myocardium during diastole is positive (the intraventricular pressures are greater than the intrapericardial pressures), thus facilitating ventricular filling. The accumulation of fluids

in the pericardial cavity causes an increase of both the intrapericardial and intracardial pressures, but the difference between them narrows, reducing the distending force for ventricular filling. Even in cases of diffuse circumferential effusion, the hemodynamic effects of tamponade are due primarily to right heart compression; in fact, the pressures of these thinnerchambers equilibrate with rising pericardial pressure before the left atrial and ventricular pressures. With overt tamponade, rising of the pericardial pressure progressively reduces and ultimately makes phasically negative the transmural pressure of first the right and then the left heart chambers. Filling of the heart is therefore maintained by a parallel increase in systemic and pulmonary venous pressures; as compensatory mechanisms (primarily tachycardia) are defeated, cardiac filling decreases, pericardial pressure equilibrates with left ventricular (LV) diastolic pressure, and cardiac output decreases critically. Inspiration increases the filling gradient across the right heart but not the left heart; the augmented right ventricular (RV)

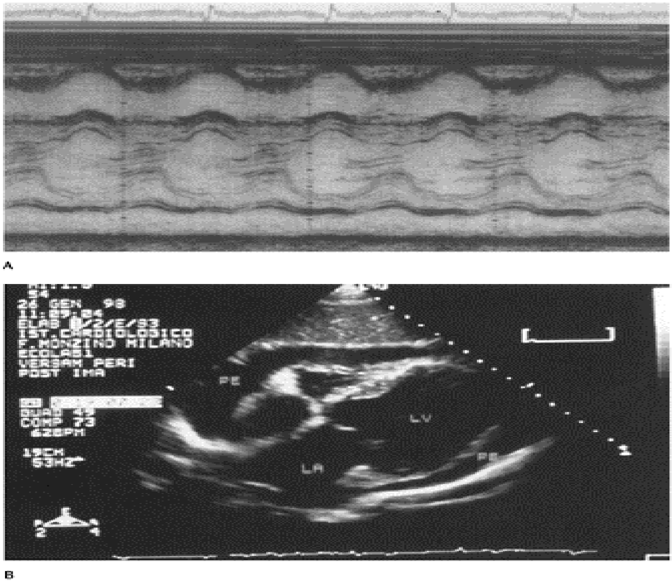


Figure 2.1 M-mode (A), subcostal four-chamber (B), parasternal short-axis (C), and posterior view (from a back transducer location) (D) of patients with pericardial effusion. The posterior view allows us to demonstrate the association with a left pleural effusion. PE: pericardial effusion; LV: left ventricle; RV: right ventricle; LA: left atrium; Ao: aorta.

filling occurs at the expense of reduced filling and stroke output of the left ventricle (LV); these mechanisms cause a typical clinical sign of tamponade, pulsus paradoxus (systolic drop in arterial pressure of 10 mmHg or more during normal breathing).[1–3]

Clinical signs

Although the clinical conditions may be markedly various in patients with cardiac tamponade (due mainly to different etiologies of the syndrome), three signs (Beck's triad), hypotension, jugular venus distension (elevated systemic venous pressure), and pulsus paradoxus, are typically associated with cardiac tamponade. Tachycardia, dyspnea, cough and dysphagia, chest discomfort, shock, and unconsciousness may also be present. However, rapid bleeding or fluid accumulation in the pericardial sac may present with more overt signs and symptoms than in patients who have more gradual onset of the syndrome and preserved blood pressure.

three signs, in particular the pulsus paradoxus, may be Atypical clinical presentation is very frequent, and the absent in several settings: LV dysfunction, regional right atrial (RA) tamponade, positive-pressure breathing, atrial septal defect, pulmonary arterial obstruction, and severe aortic regurgitation.

Echocardiographic Doppler features of cardiac tamponade

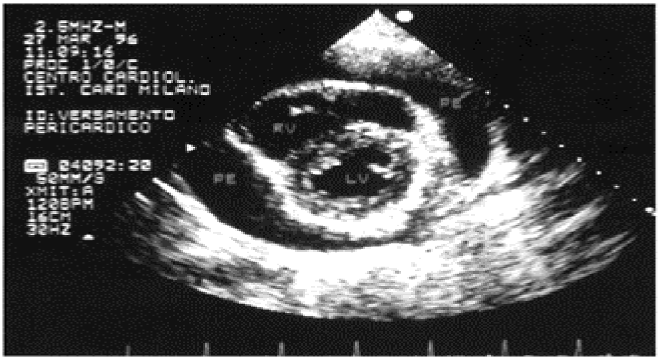
Pericardial effusion is easily demonstrated by echocardiography; when tamponade is suspected, the evaluation of the pericardial sac should be carefully performed through all the echocardiographic windows, in order to quantitate pericardial fluid and distinguish diffuse circumferential effusions from loculated regional ones. Echocardiography allows discrimination between small, moderate, and large effusions and may also detect the association with pleural effusions.[4–7] In this regard, Figure 2.1 shows examples of pericardial effusions analyzed by different approaches: parasternal M-mode, subcostal two-dimensional view, parasternal short-axis view, and one case in which the association with pleural effusion is well documented by a posterior view (from the back of the patient).

The assessment of the character of the fluid by ultrasound is not possible since serous effusions, hemopericardium, and chilopericardium all appear as similar clear spaces. However, fibrous strands are frequently observed in cases of chronic diseases, and hematomas and clots may be detected as solid masses of granular echoes inside the pericardial sac. Echogenic masses attached to the visceral pericardium may suggest the presense of metastasis.

Excessive cardiac motion up to the 'swinging heart' is frequently seen in severe pericardial effusion with chronically accumulated effusion and a minimum of adhesions. This movement has been observed in malignancies, chronic tuberculous pericarditis, and benign viral pericarditis.

Table 2.1 lists the echocardiographic and Doppler signs that have been described in cardiac tamponade.

An exaggerated inspiratory expansion of the right ventricle (RV) and simultaneous compression of the LV are a nonspecific sign of increased direct interdependence, and this has therefore a low specificity. Diastolic collapse of the RA and RV free walls are accepted signs of cardiac tamponade; they can be evaluated by both M-mode and two-dimensional echocardiography.[8–10] Figure 2.2 shows



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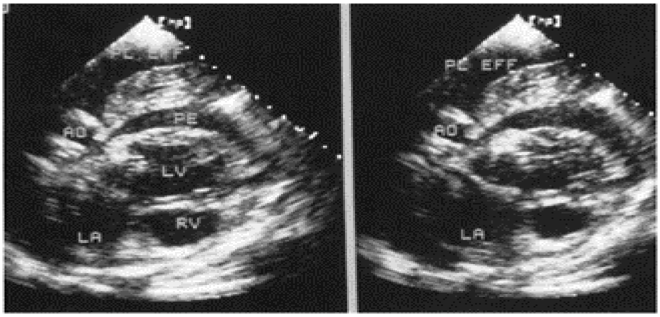

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Table 2.1 Echo-Doppler signs of cardiac tamponade

Exaggerated inspiratory variation of the two ventricles (inspiratory expansion of the RV and simultaneous compression of the LV; reciprocal changes in the expiratory phase)
Right atrial collapse
Right ventricular collapse
Left atrial collapse
Left ventricular collapse
Inferior vena cava plethora
Abnormal increased respiratory variation in transvalvular blood flow velocities (mitral and aortic flow reduction in the inspiratory phase)

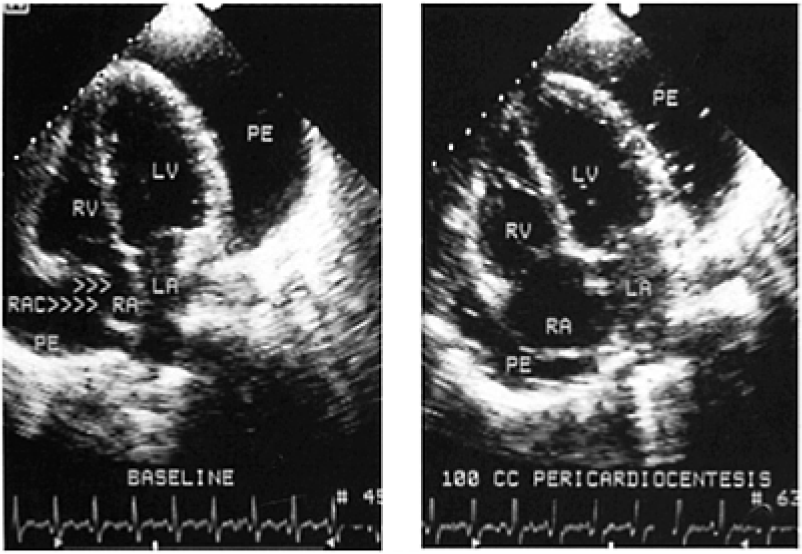

Figure 2.2 Apical four-chamber echocardiogram of a patient with pericardial effusion causing cardiac tamponade. Arrows indicate the presence of right atrial collapse (RAC) (left panel), which disappears immediately after 100-ml pericardiocentesis (right panel). PE: pericardial effusion; LV: left ventricle; RV: right ventricle; LA: left atrium; RA: right atrium.

these two signs in the same patient. RV collapse is a transient invagination of the RV free wall which occurs in early diastole, while RA collapse is a transient invagination of this wall that occurs in late diastole and early systole. Timing of these two collapses is related to the lowest intracavitary pressures occurring in the two chambers in early (RV) or late (RA) diastole, respectively. These two signs of tamponade may be too sensitive on the one hand and lack specificity on the other. Therefore, the duration of the collapse should be taken into account. In fact, the duration of collapses is directly related to severity of tamponade, and it improves the specificity and predictive value of these diagnostic signs.[11] With increasing severity, RA collapse tends to begin earlier and RV collapse to extend later in diastole. Even though these two signs are too sensitive, the presence of both RA and RV collapse always indicates that the effusion is hemodynamically significant. In accordance with this concept, Figure 2.3 shows a patient with cardiac tamponade. The image recorded immediately before pericardiocentesis demonstrates the presence of RA collapse; this sign disappeared immediately after 100

ml pericardial drainage (even though the amount of pericardial effusion was still very important). In an experimental animal study, Leimgruber et al[12] showed that the earliest appearance of RV

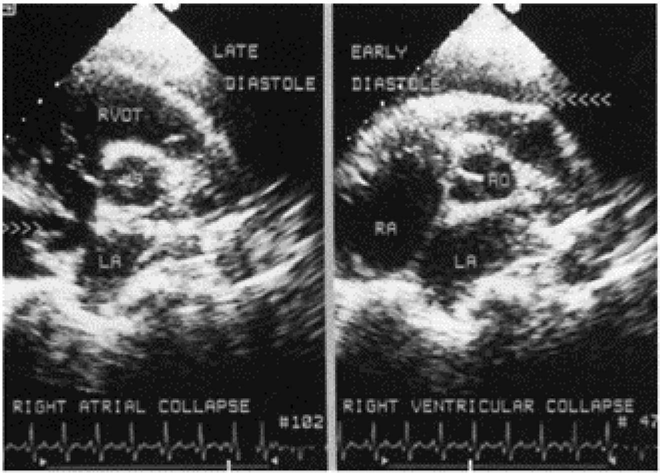

Figure 2.3 Short-axis, two-dimensional echocardiogram through the base of the heart in a patient with pericardial effusion and cardiac tamponade. Late diastolic right atrial collapse (left panel) and early diastolic right ventricular collapse (right panel) are noted as indentation of the two walls. Ao: aorta; RA: right atrium; LA: left atrium; RVOT: right ventricular outflow tract.

diastolic collapse is associated with a 21% reduction in cardiac output at a time when mean aortic pressure is unchanged. Lopez-Sendon et al[13] in an open-chest dog model showed the sequence and characteristics of RA collapse: long before hemodynamic alterations of fully established cardiac tampoande are present, RA compression becomes apparent as a quick inward motion of a small portion of the posterior RA wall, and as the intrapericardial pressure increases, a wider portion of the RA wall presents an abnormal motion. With a further increase in intrapericardial pressure, RA inversion throughout the entire cardiac cycle becomes progressively apparent, and finally the complete distortion of the RA shape and dimensions indicates an extreme situation of cardiac tamponade. These old experimental studies underline the concept that echocardiography may detect early tamponade and the hemodynamic significance of pericardial effusion, and that we may always consider tamponade as a continuum of events: in the early phase of cardiac

compression, even minor elevations of intrapericardial pressure produce some effects on ventricular filling, and echocardiographic signs could be present even in the absence of overt clinical tamponade.[14–18] There are few exceptions to the use of right-sided collapse for diagnosing tamponade: RV hypertrophy and high RV intracavitary pressures may prevent the occurrence of these collapses.

Left atrial and LV collapse are rarely seen in patients with cardiac tamponade. This is mainly due to local factors; first of all, the LV is much thicker and stiffer than the other chambers and for that reason it resists collapse. The left atrium is posteriorly positioned, and it is tightly clasped by the pericardium; rarely, in cases with very large effusions, does fluid get behind the left atrium and cause wall collapse. Facilitating factors of left atrial and ventricular wall collapse are therefore posteriorly loculated effusions and conditions in which pressures in these chambers are relatively low. LV collapse has been described in postoperative effusion;[19] Figure 2.4 shows a patient who had a large, loculated postoperative effusion

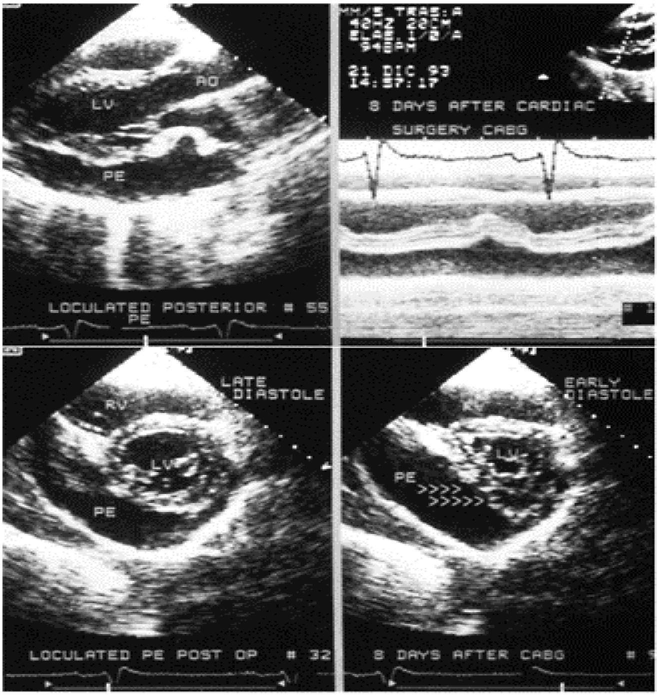

Figure 2.4 Parasternal long axis (top left), M-mode tracing (top right), and parasternal short axis in late diastole (bottom left) and in early systole (bottom right) in a patient with diastolic collapse of the left ventricle

due to a posteriorly loculated pericardial effusion occurring after cardiac surgery. PE: pericardial effusion; LV: left ventricle; Ao: aorta.

(6 days after coronary artery bypass grafting) causing cardiac tamponade. By both M-mode and two-dimensional echocardiography, the LV posterior wall exhibits an invagination during early diastole.

Inferior vena cava (IVC) plethora with reduced inspiratory collapse is another sign of cardiac tamponade;[20] several studies have shown that dilation, particularly blunted inspiratory variation of the inferior vena cava, correlates with increased right atrial pressure. Therefore, this is a sensitive but unspecific sign of the syndrome, which may be seen also in constrictive pericarditis, RV infarction, pulmonary hypertension, and tricuspid regurgitation. Through this method (inspiratory variation during regular respiration), RA pressure can be estimated and classified as normal (6 mmHg in the presence of an inspiratory reduction of the IVC diameter by over 45%), moderately elevated (9 mmHg; 35–45%), or markedly elevated (16 mmHg; <35%).[21]

Cardiac tamponade is associated with an abnormally increased respiratory variation in transvalvular blood flow velocities.[22,23] Normally, inspiration causes a minimal increase in systemic venous, tricuspid, and pulmonary valvular blood flow and a corresponding decrease in pulmonary venous, mitral, and aortic flow velocities.

Thus, in a normal subject, inspiratory variations in these measurements are less than 20%. With cardiac tamponade, ventricular interdependence is exaggerated, and inspiration produces a significant decrease in left-sided filling. Accordingly, mitral and aortic valvular flows are reduced by more than 40% (and a corresponding increase is observed in right-sided flow velocities). Figure 2.5 demonstrates the usefulness of Doppler echocardiography point of view, it is important to control the positioning of in the setting of cardiac tamponade. From a technical the sample volume during the examination by trying to exclude differences in location during respiration, and to utilize the EGG cable as respiratory monitoring (several ultrasound devices have this option).

Pericardial effusion and cardiac tamponade after cardiac surgery

Pericardial effusion is not a rare complication of cardiac surgery.[24–28] Although it is generally reversible and not life-threatening, it may sometimes evolve toward cardiac tamponade. The role of echocardiography is extremely important in the diagnosis of cardiac tamponade in the postoperative period, since the clinical and imaging presentations could be atypical; therefore, a specific knowledge of this disorder is very useful.[29]

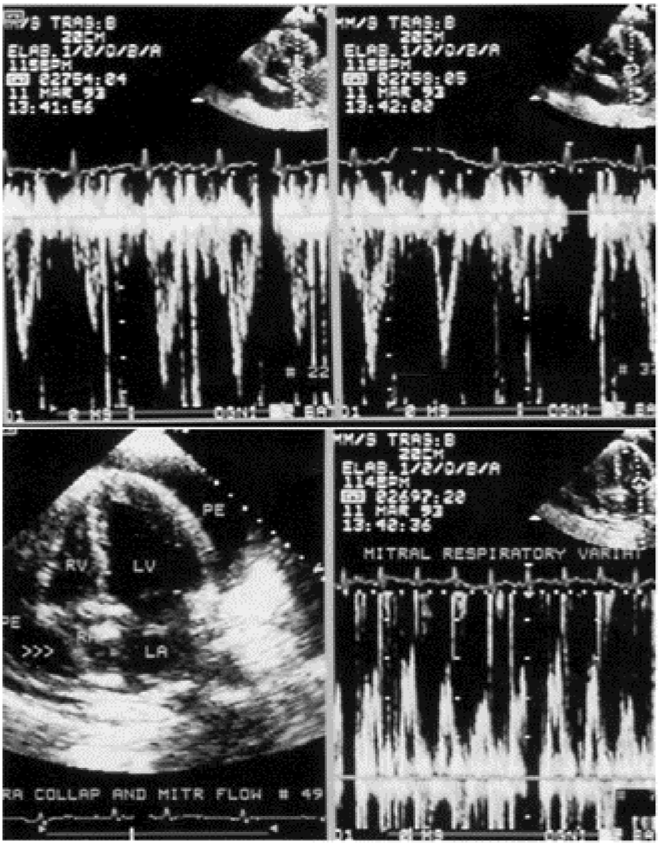

Figure 2.5 Aortic flow (top panels) and mitral flow (bottom right) changes during respiratory phases in a patient with cardiac tamponade. Inspiration produces a marked decrease in left-sided filling (both aortic and mitral flow). In the same patient, right atrial collapse (bottom left: four-chamber apical view) is present. PE: pericardial effusion; LA: left atrium; RA: right atrium; LV left ventricle; RV: right ventricle.

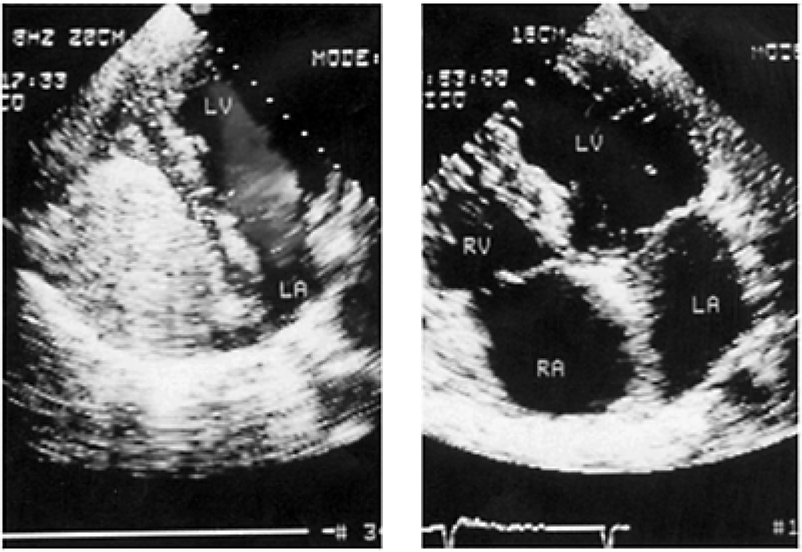

Figure 2.6 Left panel: apical four-chamber view showing an echo-dense voluminous mass (pericardial clot) at the level of the right atrium. Due to this compression, the right atrial cavity is virtual. Right panel: after subxiphoid pericardial drainage, the pericardial clot has been completely removed, and the size of the right atrium is normal. LA: left atrium; LV left ventricle; RA: right atrium; RV: right ventricle.

Virtually all pericardial effusion is found by postoperative day 5; it peaks on day 10 and resolves within 1 month. In a large, prospective study,[30] pericardial effusion was detected in 64% of cases and was more often associated with coronary artery bypass grafting (75%) than with valve replacement (52%) or other types of surgery (50%). It was small in 68%, moderate in 30%, and large in 2% of cases. Loculated effusions (57.8%) were more frequent than diffuse ones (42.2%). The size and site of effusions were related to the type of surgery; in particular, small pericardial effusion was slightly more frequent after valve replacement than after coronary artery bypass grafting. After valve replacement, diffuse fluid accumulations were more frequent (55%) than loculated ones, whereas after coronary artery bypass grafting, loculated effusions were more common (63.5%). In particular, anterior loculated effusions were more common after coronary artery bypass grafting, and posterolateral ones after valve replacement. Interestingly, 6% of the patients had isolated effusion along the RA wall; this type of effusion can compress the heart and can be difficult to diagnose in patients with postoperative low

output failure. Fifteen out of the series of 780 patients had cardiac tamponade; this event was significantly more common after valve replacement than after coronary artery bypass grafting or other types of surgery. All these epidemiologic data are extremely important, since the recognition of cardiac tamponade in the early or late postoperative periods is not easy.

Loculated effusions may be difficult to explore, and off-axis views should be attempted in cases in which the differential diagnosis with pleural effusion is difficult or in the presence of atypical presentation. In particular, localized pericardial effusion or clot at the level of the RA wall, RV, LV, and left atrium may occur after cardiac surgery and present with unique clinical and echo-Doppler features. Modified apical, subcostal, and posterior views are extremely useful in the evaluation of all these isolated effusions; however, in several cases, particularly when transthoracic echocardiography does not provide complete imaging of the pericardial sac and unstable hemodynamics coexists with suspected cardiac tamponade, transesophageal echocardiography is mandatory.[31,32] Figure 2.6 shows a transthoracic example of a pericardial clot compressing the right atrium diagnosed through transthoracic echocardiography; thrombosis of this loculated pericardial effusion after aortic valve replacement mimicked a RA mass.[33] A transesophageal example of pericardial hematoma compressing the left atrium is shown in Figure 2.7. In both cases, patients were in shock, and emergency echocardiography was requested. Echocardiography allowed rapid diagnosis; immediate surgical decompression was performed successfully with clinical resolution of shock. These two cases reinforce the concept of the usefulness of echocardiography in unstable patients after cardiac surgery.[34] Russo et al[29] demonstrated that 9 out of 10 patients with cardiac tamponade (10/510 consecutive patients evaluated after cardiac surgery) had either atypical clinical, hemodynamic, and/or echocardiographic findings. The authors found that patients frequently had vague and nonspecific initial symptoms, including general malaise, lethargy, anorexia, and palpitations, whereas dyspnea, chest pain, confusion, diaphoresis, and oliguria occur generally later. Selective chamber compression was the more frequent cause of atypical clinical and echo presentation. LV and RV, and left and right atrial compressions have been demonstrated after cardiac surgery. Pericardial clots, particularly at the level of the right chambers, may produce low cardiac output soon after open-heart surgery with hemodynamic signs very similar to constrictive pericarditis; Beppu et al[32] showed the indispensability of transesophageal echocardiography in these cases. Several reports confirmed that after cardiac surgery and tamponade caused by loculated regional RA compression, pulsus paradoxus may be absent, while regional compression of the LV and tamponade may occur despite normal RA and peripheral venous pressure.

In conclusion, atypical clinical presentation is very frequent after cardiac surgery, and the three clinical signs of tamponade (in particular, the pulsus paradoxus) may be absent in several settings: LV dysfunction, regional RA tamponade, positive-pressure breathing, atrial septal defect, pulmonary arterial obstruction, and severe aortic regurgitation. In terms of hemodynamic effects, Fowler et al[35] demonstrated that right-sided cardiac compression has more important effects than does left-sided compression; however, left-sided tamponade still makes a significant contribution to the total hemodynamic picture of cardiac tamponade. Therefore, echocardiographic research of regional pericardial effusion should be very accurate. Postoperative cardiac tamponade might be suspected in

the presence of loculated effusions along the RA and/or along its junction with the superior vena cava, left atrial or bilateral atrial compression, and RV or LV effusions. In this setting, we investigated the type and frequency of Doppler and echocardiographic findings of cardiac tamponade.[30] We invariably (15/15 cases in our series) recorded an inspiratory decrease (>25%) in the velocity of flow through the aortic and mitral valves; five patients had RA collapse, RV collapse, and inferior vena cava plethora; eight had two of these signs, and two had one. In four patients, moderate pericardial effusions were associated with one or two echocardiographic signs of cardiac tamponade, but not with clinical evidence of hemodynamic embarrassment or with Doppler signs of tamponade.

Echo-guided pericardiocentesis

Early recognition and treatment of patients with suspected tamponade represent an important clinical goal, and once it is diagnosed, decompression by a safe, simple, and rapid method is required.

Drainage of an acute, effusion-producing cardiac tamponade may be performed by two alternative techniques: subxiphoid pericardiotomy and percutaneous pericardiocentesis. Subxiphoid pericardiotomy[36] is performed under either local or general anesthesia via a midline longitudinal incision from the xiphosternal junction to 6–8 cm below the tip of the xiphoid. The xiphosternal point is then split, and the xiphoid process removed to expose the subxiphoid diaphragm. The anterior diaphragm is depressed and the sternum can be lifted to allow dissection and removal of fatty tissue until the pericardium is well reached; then a finger is introduced to palpate the tension of the pericardial sac, and the pericardium is grasped with forceps and incised, allowing the fluid to escape. Samples of the pericardial effusion can be collected in sterile tubes and sent for bacteriologic analysis, and finally a pericardial drain may be left in place for 24 h or more.

Percutaneous pericardiocentesis has been described in detail by several authors,[37–41] who proposed improvement of the technique, starting from the blind procedure, and passing through echo-guided and contrast echo-guided pericardiocentesis. Callaghan et al[39] emphasized visualization of the pericardial needle during their study on echo

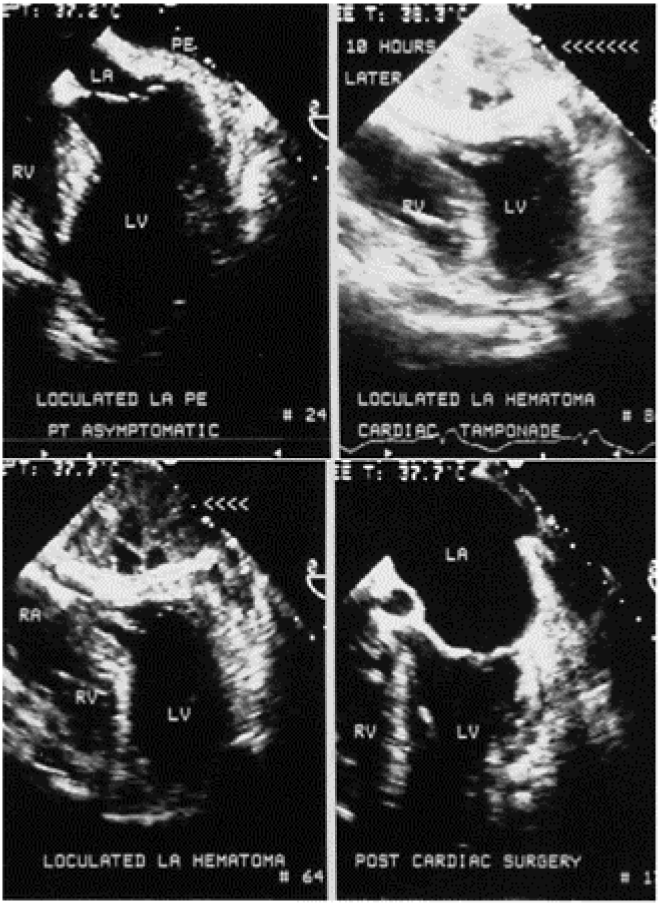

Figure 2.7 Transesophageal four-chamber view (top, left panel) in a patient with hemodynamic instability immediately after a coronary artery by-pass grafting. A moderate pericardial effusion along the left atrium was demonstrated, but the presence of severe left ventricular dysfunction led to the decision to position an intraaortic balloon, starting the counterpulsation and optimizing the medical therapy. A few hours later, the transesophageal echocardiography was

repeated because of severe cardiogenic shock; a large pericardial hematoma compressing the left atrium was clearly demonstrated (top right panel and bottom left panel). Therefore, the patient underwent immediate surgical drainage of the pericardium with resolution of the clinical and hemodynamic instability and normalization of the left atrial cavity (bottom right panel). LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle; PE: pericardial effusion.

guided pericardiocentesis with the assistance of contrast echocardiography, thus reducing the likelihood of heart puncture. An improvement of ultrasonically guided pericardiocentesis has been proposed by our group,[40] with the specific purpose of facilitating posterior drainage. The Tuohy needle utilized in this study, thanks to its curved tip, greatly facilitated guidance of the wire and catheter to the posterior pericardial space, so that the standard Seldinger technique was successful not only in massive and diffused effusions, but also in loculated posterior ones. More recently, we compared the combined use of two-dimensional echo monitoring by the described technique with surgical subxiphoid pericardiotomy in the treatment of acute cardiac tamponade due to pericardial effusion occurring after cardiac surgery.[41] Forty-two patients were included in the study: during the first period, one of the two methods was chosen by the clinical staff, whereas in the second period percutaneous pericardiocentesis was the treatment of choice. Complete drainage of pericardial fluid by percutaneous pericardiocentesis was obtained in 26 out of 29 patients (86%). No major complication occurred with the use of the two techniques. In three cases, in the second study period, the minimal amount of fluid or pericardial hematoma indicated surgical approach as the first therapeutic choice; percutaneous pericardiocentesis was unsuccessful in three cases without complications, and surgical drainage was therefore performed. This study suggested that the more invasive technique (subxiphoid surgical pericardiotomy) should be selected in cases in which percutaneous pericardiocentesis is unsuccessful or when the echocardiographic examination discourages a percutaneous approach. Figure 2.8 shows how echo-guided pericardiocentesis can assist in the drainage of the pericardial sac.

Tsang et al[42] reported the Mayo Clinic experience in cardiac tamponade caused by cardiac perforation as a complication of interventional catheterization. Echocardiography-guided pericardiocentesis was safe and effective in rescuing patients from tamponade and reversing hemodynamic instability complicating invasive cardiacbased procedures. In this clinical setting as well as in cardiac tamponade due to

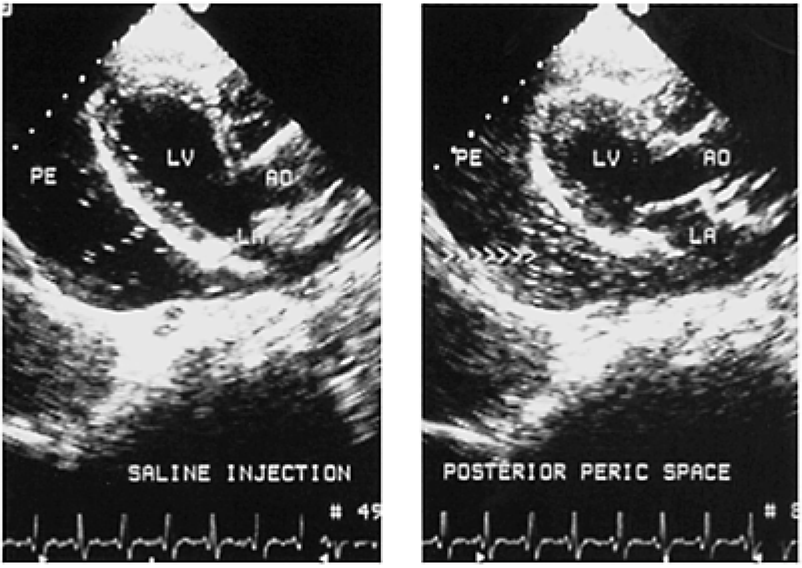

Figure 2.8 Echo-guided pericardiocentesis. The long-axis, parasternal view shows the presence of a large pericardial effusion. In the left panel and more markedly in the right panel, this view illustrates echogenic microbubbles inside the posterior pericardium after saline injection through the needle, thus confirming the correct positioning of the needle in the pericardial space. PE: pericardial effusion; Ao: aorta; LV: left ventricle; LA: left atrium.

other etiologies, they proposed a specific technique based on the two-dimensional characteristics of effusions. Two-dimensional echocardiography allowed the examiner to localize the largest collection of pericardial fluid in closest proximity to the transducer, thereby identifying the ideal entry site. The trajectory of the pericardiocentesis needle was defined by transducer angulation. After local infiltration with 1% lidocaine, a Polytef-sheathed needle (16–18 gauge) was used for the initial pericardial entry. On reaching the pericardial fluid, the steel core of the needle was immediately withdrawn, leaving only the Polytef sheath in the pericardial space. In the presence of hemorrhagic effusion, injection of a small volume of agitated saline as a contrast was used for echocardiographic confirmation of the sheath position. The sheath was withdrawn, and a standard dilator and introducer (6–7F) were advanced over the guide wire. A pigtail

angiographic catheter (65 cm; 6–7F) was then introduced into the pericardial space. Rescue echo-guided pericardiocentesis by the described technique was the only treatment required for the majority of these patients. Surgical exploration or intervention is indicated when complete control of the bleeding is in question or when hemodynamic instability is not rapidly restored by rescue pericardiocentesis alone.

Many investigators have suggested two-dimensional echocardiography to guide not only the placement of the pericardiocentesis needle, but also biopsy forceps, balloon catheters (pericardioplasty), and other new percutaneous devices.[43,44] Instillation in the pericardial sac of various agents with sclerosing, anti-inflammmatory or cytostatic activity has been also proposed under echocardiographic assistance.

Conclusions

Echocardiography aids in the detection, localization, and quantitation of pericardial effusion. Various echocardiographic modalities including M-mode, two-dimensional transthoracic, and transesophageal echocardiography, have the greatest application in assessing cardiac tamponade as well as in the management of the syndrome. The role of ultrasound techniques is very important not only in the presence of large and diffuse pericardial effusion and classic clinical presentation, but also in patients with atypical clinical presentation and/or loculated pericardial effusion. The value of using echocardiography to assist in monitoring pericardiocentesis has been demonstrated.

References

1. Spodick D. Pericardial disease. In: Braunwald, Libby and Zipes, eds, Heart Disease, 6th edn. Philadelphia: Saunders, 2001; 1823–76.
2. Brockington G, Schwartz S, Pandian N, eds. Echocardiography in pericardial diseases. In: Cardiac Imaging (a Companion to Brauwald's Textbook). Philadelphia: Saunders, 1991.
3. Spodick DH. Pathophysiology of cardiac tamponade. Review. Chest 1998; 113:1372–8.
4. Feigenbaum H. Echocardiography. Philadelphia: Lea and Febiger, 1994.
5. Parameswaran R, Goldberg H. Echocardiographic quantitation of pericardial effusion. Chest 1983; 83:767–70.
6. Vazquez de Prada J, Jiang L, Handschumacher M, et al. Quantification of pericardial effusions by three-dimensional echocardiography. J Am Coll Cardiol 1994; 24:254–9.
7. D'Cruz I, Hoffman P. A new cross sectional echocardiographic method for estimating the volume of large pericardial effusions. Br Heart J 1991; 66:448–51.
8. Battle RW, LeWinter MM. The evaluation and management of pericardial disease. Curr Opin Cardiol 1990, 5:331–9.
9. Singh S, Wann S, Schuchard G, et al. Right ventricular and right atrial collapse in patients with cardiac tamponade—a combined echocardiographic and hemodynamic study. Circulation 1984; 70:966–71.
10. Armstrong W, Schilt B, Helper D, Dillon J, Feigenbaum H. Diastolic collapse of the right ventricle with cardiac tamponade: an echocardiographic study. Circulation 1982; 65:1491–6.
11. Reydel B, Spodick D. Frequency and significance of chamber collapses during cardiac tamponade. Am Heart J 1990; 119:1160–3.

12. Leimgruber P, Klopfenstein S, Wann S, Brooks H. The hemodynamic derangement associated with right ventricular diastolic collapse in cardiac tamponade: an experimental echocardiographic study. Circulation 1983; 68:612–20.
13. Lopez-Sendon J, Garcia-Fernandez M, Coma-Canella I, Sotillo J, Silvestre J. Mechanism of right atrial wall compression in pericardial effusion: an experimental echocardiographic study in dogs. J Cardiovasc Ultrasonogr 1988; 7:127–34.
14. Fowler N. Cardiac tamponade. A clinical or an echocardiographic diagnosis? Circulation 1993; 87:1738–41.
15. Eisenberg M, Schiller N. Bayes' theorem and the echocardiographic diagnosis of cardiac tamponade. Am J Cardiol 1991; 68:1242–4.
16. Fowler N. The significance of echocardiographic-Doppler studies in cardiac tamponade. J Am Coll Cardiol 1988; 11:1031–3.
17. Shabetai R. Changing concepts of cardiac tamponade. J Am Coll Cardiol 1988; 12:194–5.
18. Levine M, Lorell B, Diver D, Come P. Implications of echocardiographically assisted diagnosis of pericardial tamponade in contemporary medical patients: detection before hemodynamic embarrassment. J Am Coll Cardiol 1991; 17:59–65.
19. Chuttani K, Pandian N, Mohanty PK, et al. Left ventricular diastolic collapse. An echocardiographic sign of regional cardiac tamponade. Circulation 1991; 83:1999–2006.
20. Himelman R, Kircher B, Rockey D, Schiller N. Inferior vena cava plethora with blunted respiratory response: a sensitive echocardiographic sign of cardiac tamponade. J Am Coll Cardiol 1988; 12:1470–7.
21. Pepi M, Tamborini G, Galli C, et al. A new formula for echo-Doppler estimation of right ventricular systolic pressure. J Am Soc Echocardiogr 1994; 7:20–6.
22. Appleton C, Hatle L, Popp R. Cardiac tamponade and pericardial effusion: respiratory variation in transvalvular flow velocities studied by Doppler echocardiography. J Am Coll Cardiol 1988; 11:1020–30.
23. Schutzman J, Obarski T, Pearce G, Klein A. Comparison of Doppler and two-dimensional echocardiography for assessment of pericardial effusion. Am J Cardiol 1992; 70:1353–7.
24. Miller R, Horneffer P, Gardner T, Rykiel M, Pearson T. The epidemiology of the postpericardiotomy syndrome: a common complication of cardiac surgery. Am Heart J 1988; 116:1323–9.
25. Weitzman L, Tinker WP, Kronzon I, Cohen M, Glassman E, Spencer F. The incidence and natural history of pericardial effusion after cardiac surgery—an echocardiographic study. Circulation 1984; 69:506–11.
26. Ofori-Krakie S, Tuberg T, Geha A, Hammond G, Cohen L, Langou R. Late cardiac tamponade after open heart surgery: incidence, role of anticoagulants in its pathogenesis and its relationship to the postpericardiotomy syndrome. Circulation 1981; 63:1323–8.
27. Bommer W, Follette D, Pollock M, Arena F, Bognar M, Berkoff H. Tamponade in patients undergoing cardiac surgery: a clinical-echocardiographic diagnosis. Am Heart J 1995; 130:1216–23.
28. Ikaheimo M, Huikuri H, Airaksinen J, et al. Pericardial effusion after cardiac surgery: incidence, relation to the type of surgery, antithrombotic therapy, and early coronary bypass graft patency. Am Heart J 1988; 116:97–102.
29. Russo A, O'Connor W, Waxman H. Atypical presentations and echocardiographic findings in patients with cardiac tamponade occurring early and late after cardiac surgery. Chest 1993; 104:71–8.
30. Pepi M, Muratori M, Barbier P, et al. Pericardial effusion after cardiac surgery: incidence, site, size, and haemodynamic consequences. Br Heart J 1994; 72:327–31.
31. Saner H, Olson J, Goldenberg I, Asinger R. Isolated right atrial tamponade after open heart surgery: role of echocardiography in diagnosis and management. Cardiology 1995; 86:464–72.
32. Beppu S, Tanaka N, Nakatani S, Ikegami K, Kumon K, Miyatake K. Pericardial clot after open heart surgery: its specific localization and haemodynamics. Eur Heart J 1993; 14:230–4.

33. Pepi M, Doria E, Fiorentini C. Cardiac tamponade produced by a loculated pericardial hematoma simulating a right atrial mass. Int J Cardiol 1990; 29:383–6.
34. Shanewise J, Cheung A, Aronson S, et al. ASE/SCA Guidelines for performing a comprehensive multiplane transesophageal echocardiography examination: recommendations of the American Society of Echocardiography Council for intraoperative echocardiography and the Society of Cardiovascular Anesthesiologists Task Force for Certification of Perioperative Transesophageal Echocardiography. Anesth Analg 1999; 89:870–84.
35. Fowler N, Gabel M, Buncher R. Cardiac tamponade: a comparison of right versus left heart compression. J Am Coll Cardiol 1988; 12:187–93.
36. Alcan K, Zabetakis P, Marino N, Franzone A, Michelis M, Bruno M. Management of acute cardiac tamponade by subxiphoid pericardiotomy. JAMA 1982; 247:1143–8.
37. Goldberg B, Pollack H. Ultrasonically guided pericardiocentesis. Am J Cardiol 1973; 31:490–3.
38. Stewart J, Gott V. The use of a Seldinger wire technique for pericardiocentesis following cardiac surgery. Ann Thorac Surg 1983; 35:467–8.
39. Callahan J, Seward J, Nishimura R, et al. Two-dimensional echocardiographically guided pericardiocentesis: experience in 117 consecutive patients. Am J Cardiol 1985; 55:476–9.
40. Pepi M, Maltagliati A, Tamborini G, Susini G, Fiorentini C. Improvement in ultrasonically guided pericardiocentesis. J Cardiovasc Ultrason 1988; 7:193–6.
41. Susini G, Pepi M, Sisillo E, et al. Percutaneous pericardiocentesis versus subxiphoid pericardiotomy in cardiac tamponade due to postoperative pericardial effusion. J Cardiothorac Vasc Anesth 1993; 7:178–83.
42. Tsang T, Freeman W, Barnes M, Reeder G, Packer D, Seward J. Rescue echocardiographically guided pericardiocentesis for cardiac perforation complicating catheter-based procedures. J Am Coll Cardiol 1998; 32:1345–50.
43. Selig M. Percutaneous transcatheter pericardial interventions: aspiration, biopsy, and pericardioplasty. Am Heart J 1993; 125:269–71.
44. Ziskind A, Pearce C, Lemmon C, et al. Percutaneous balloon pericardiotomy for the treatment of cardiac tamponade and large pericardial effusions: description of technique and report of the first 50 cases. J Am Coll Cardiol 1993; 21:1–5.

3
Echocardiography in chest trauma

Gregory M Scalia and Ruth E Ramm

Key points

- Echocardiography, especially transesophageal echocardiography, is a key component of diagnostic investigations after blunt or penetrating chest trauma.
- Knowing the possible mechanisms of injuries to the heart and great vessels is crucial in interpretation of echocardiographic findings.
- Echocardiographic signs are often nonspecific and may include regional (non-coronary) distribution patterns of right and/or left ventricular dysfunction, increased myocardial wall echogenicity and thickness, signs of myocardial rupture in various locations, including atrial, septal, and free-wall rupture, with or without tamponade or pseudoaneurysm formation, acute valve regurgitation, and signs of acute aortic syndrome.
- This is an area of echocardiography which should be undertaken by the most experienced operators available.

Chest trauma, whether blunt or penetrating, may involve damage to the heart and great vessels with consequences which may range from simple hematoma formation to major organ disruption, hemodynamic collapse, and death. Chest trauma contributes over 30% of the 150 000 trauma deaths occurring annually in the USA.[1] In general, patients with significant chest trauma will be transferred to intensive care units for stabilization, monitoring, and diagnostic investigations.[2] Echocardiography, especially transesophageal echocardiography, is a key component of this investigation process. Other tests including chest radiography, electrocardiography, computed tomography (CT), and magnetic resonance imaging (MRI) scanning also have roles in the diagnostic work-up.

Trauma to the heart and great vessels is typically divided into blunt and penetrating (Table 3.1). Blunt trauma is a consequence of impact on the chest, with or without bony injury. In high-velocity blunt trauma, such as motor vehicle accidents and falls from a great height, tissue damage is a consequence of acceleration/decelera tion and shearing forces as well as the direct impact. Penetrating injury is a consequence of direct intrusion into the heart or great vessels by foreign objects. Iatrogenic injuries from medical procedures can cause such injuries. Less common causes of cardiac injury include electrocution and radiation.

Table 3.1 Cardiac trauma—classification

Blunt cardiac trauma
Myocardial contusion Cardiac rupture Valve injury Coronary injury/dissection
Great vessel trauma
Aortic transection Pseudoaneurysm Dissection
Penetrating cardiac trauma
High velocity missiles versus stabbing injuries Organ and vessel perforation, rupture, and fistula formation
Iatrogenic injuries

Blunt cardiac trauma

In the nonmilitary setting, the commonest cause of chest trauma is a motor vehicle accident. Falls from a great height, including attempted suicide, are also causes of high-velocity blunt trauma. Low-velocity injuries are seen after impact in contact sport, horse kicks, and blasts. In wmotor vehicle accidents, front-on impact causes high velocity injury, often by the steering wheel or seat belt. Side-on impact causes shear injury to the aorta.[3] Air-bag impact has been reported to rupture the right atrium.[4]

Myocardial contusion

Myocardial contusion involves myocyte damage usually with intramural hematoma/bruising.[5] Because of its anterior location, the right ventricle is the most vulnerable to this form of injury. The anteroapical portion of the left ventricle is also vulnerable, particularly to left lateral impact. Ventricular arrhythmia[6] and conduction disturbances with right bundle branch block have been described.[7] At echocardiography, right or left ventricular dysfunction may be seen, often in a regional (non-coronary) distribution.[8] The contused area may have increased echogenicity and increased thickness, and there may be associated chamber dilation (particularly in right ventricular trauma).[9]

Cardiac rupture

Traumatic rupture of the heart is a common autopsy finding in fatal blunt chest injury cases. Atrial rupture is more common than ventricular rupture.[10] Generally, these patients

suffer tamponade with massive hemodynamic collapse. In rare cases, traumatic ventricular or atrial septal rupture can occur (Figure 3.1).[11] Pericardial tears are often associated with blunt myocardial trauma, leading to intrapericardial bleeding and effusion.[12]

Free rupture of the heart is associated with massive tamponade and hemodynamic collapse.[13] The findings at echocardiography will be as seen in tamponade cases (see Chapters 2 and 4). In some cases, the rupture is contained by a pseudoaneurysm membrane. In this situation, a blood-filled cavity in communication with the ventricle will be present. To-and-fro flow into this cavity will be evident, and thrombus may form within the false aneurysm. This finding at echocardiography should prompt the treating team to consider urgent cardiac surgery, as such pseudo-aneurysms are highly unstable. Complete rupture with tamponade can occur abruptly with drastic consequences.

Valve Injury

tricuspid valves in that order of frequency, with acute Blunt injury can cause trauma to aortic, mitral, and regurgitation being the usual consequence (see Chapters 1 and 13). Aortic valve trauma usually is manifested as

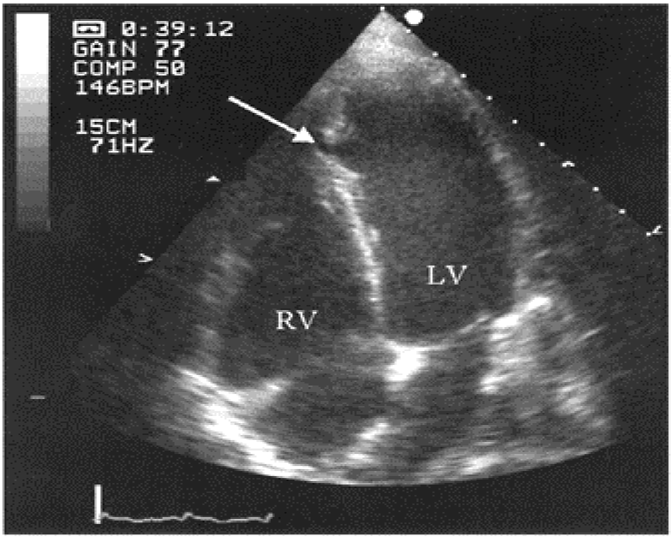

Figure 3.1 Traumatic apical ventricular septal defect (arrow) after blunt chest impact in a motor vehicle accident (LV: left ventricle; RV: right ventricle).

tears along the coaptation surface of the cusps near to the commissures. Mitral trauma may cause chordal or papillary muscle avulsion, or, more rarely, tearing or perforation of the leaflets themselves.[14] Tricuspid valve trauma usually involves damage to and/or avulsion of the anterior leaflet.[15,16] The pulmonary valve is seldom involved.

Coronary injury/dissection

Although rare, coronary artery trauma,[17] thrombosis,[18] or dissection[19] can be consequences of thoracic trauma.[20] Echocardiography may offer important clues to these possibilities by identifying regional wall motion abnormalities of the left ventricle with or without right ventricular dysfunction (see Chapter 14).[21] Such findings may guide the treating team toward coronary angiography as a matter of urgency.

Great vessel trauma

Anatomy and injury mechanisms

Blunt trauma to the chest, especially high-velocity injuries caused by motor vehicle accidents, causes shearing-type injuries of the great vessels; in particular, rupture or avulsion of the aorta. One in six patients who die of blunt chest trauma from motor vehicle accidents do so because of traumatic aortic disruption or transection.[22] Probably less than 20% of patients with aortic transection survive to hospital.[23]

The arch of the aorta is fixed by the head and neck vessels superiorly, and the descending aorta is fixed against the posterior wall of the thorax. Vertical deceleration injuries therefore tend to cause tears at the base of the innominate artery. As noted below, this area is poorly visualized by transesophageal and transthoracic echocardiography. The surgical approach to trauma to this area of the aorta is via median sternotomy. Horizontal shear injuries, which are commonly seen in motor vehicle accidents, typically cause tearing at the point of attachment of the ligamentum arteriosus, just distal to the left subclavian artery, where the arch joins the descending aorta. The surgical approach for this type of trauma is via lateral thoracotomy.

Because of the potentially lethal and legal consequences of a missed diagnosis, any diagnostic method that is used must be extremely sensitive in detecting injuries to the thoracic aorta and its branches. Moreover, because a false-positive test could lead to an unnecessary thoracotomy in an already ill patient, the avoidance of false-positive results is equally important. Thus, the diagnostic studies together must either confirm or exclude the diagnosis of aortic disruption with an exceptional degree of certainty. There must be ‘zero tolerance’ of inaccuracy.[24]

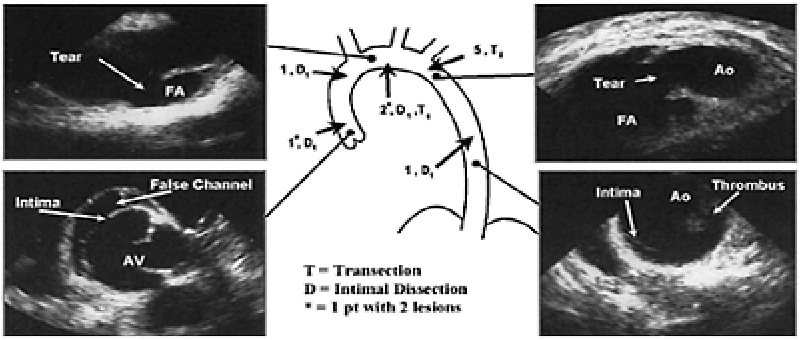

Figure 3.2 Clinical experience in a major cardiac trauma referral center with aortic trauma. Aortic transection and dissection are most common in the distal arch, but may be seen in the ascending aorta, midarch, and descending aortic areas. AV, aortic valve; Ao, aorta; FA, false aneurysm.

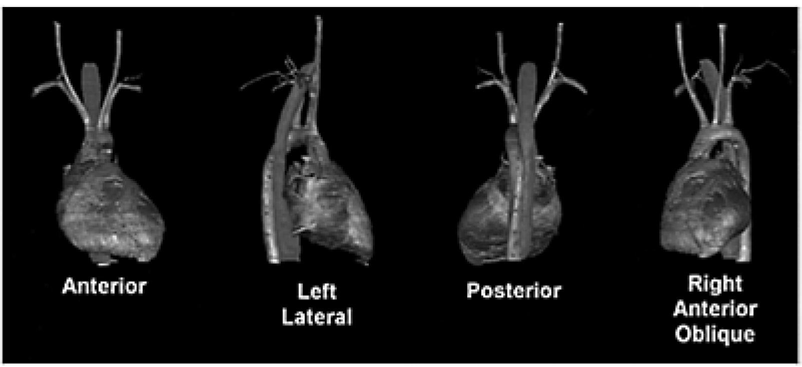

Figure 3.3 Anatomical relationship of the esophagus (pink) with the heart and aorta. The distal ascending aorta is clearly in the far field from the transesophageal probe, whereas the distal arch is a very near-field structure. (Images created with the Visible Human Dissector, Touch of Life Technologies, Colorado, USA.)

Echocardiogmphic findings

Shear injuries of the aorta can result in a variety of pathologic configurations. Complete rupture or transaction of the aorta with free intrathoracic bleeding results in rapid demise in 70–90% of cases.[25] Cases of rupture which reach hospital and undergo imaging tend to have so-called contained rupture, where the bleeding has been walled off by a pseudomembrane.[26] This configuration, like ventricular pseudoaneurysm, has inherent instability with the ever-present possibility of rapid deterioration if the containment fails (Figure 3.2[27]).

At echocardiography, contained rupture of the aorta is best visualized from the high transesophageal view (Figure from the descending aorta is seen, with torn and mobile 3.3). Discontinuity/transaction of the arch of the aorta ends often visible. There is usually perivascular hematoma,[28] with or without to-and-fro flow of liquid blood into the pseudoaneurysm space.[26] Transesophageal echocardiography in the hands of experienced operators is highly sensitive and specific for the detection of this form of aortic injury.[29] In some cases, there can be obstruction to flow across the transaction, and gradients can be detected by Doppler.

Blunt aortic trauma may also cause intimal tearing, like spontaneous dissection or intramural hematoma.[30,31] This configuration tends to have more inherent stability with a more benign course. Echocardiographic appearances are similar to those seen in spontaneous dissection (see Chapter 12).[32] Again, transesophageal views are most useful (Figure 3.4). The site of the tear should be carefully examined for perivascular hematoma and for patency of the entries of the head and neck vessels.

It is extremely important to be aware that the blind spot for transesophageal echocardiography caused by the trachea and the right main bronchus can be a source of false-negative examination results for aortic trauma. If the suspicion of aortic trauma is high, particularly if there is mediastinal hematoma, a second imaging modality, such as angiography or CT scanning, is indicated.[33]

Penetrating cardiac trauma

Anatomical and mechanical considerations

Heart and surface relationships

The heart is located within the middle mediastinum, and is protected by the bony structures of the sternum and the third and fourth ribs (Figure 3.5). The heart is bordered anteriorly by the right ventricle, laterally by the left ventricle, superiorly by the great vessels of the aorta and pulmonary artery, and inferiorly by the veins of the vena

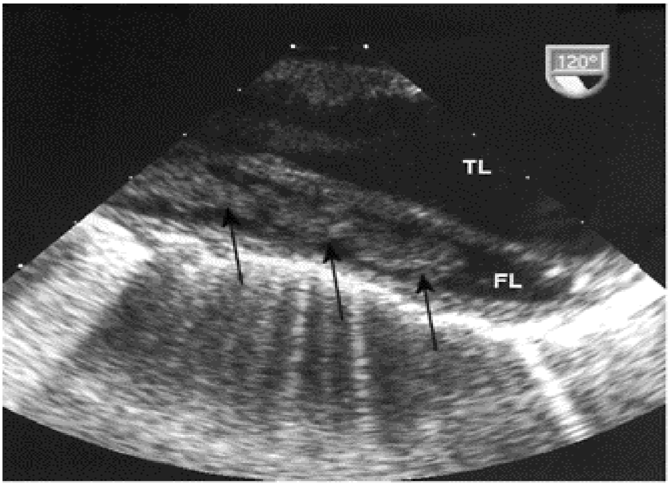

Figure 3.4 Traumatic aortic dissection after motor vehicle accident in young adult. Note the formed thrombus (black arrows) floating in the false lumen (FL). There is normal flow in the true lumen (TL).

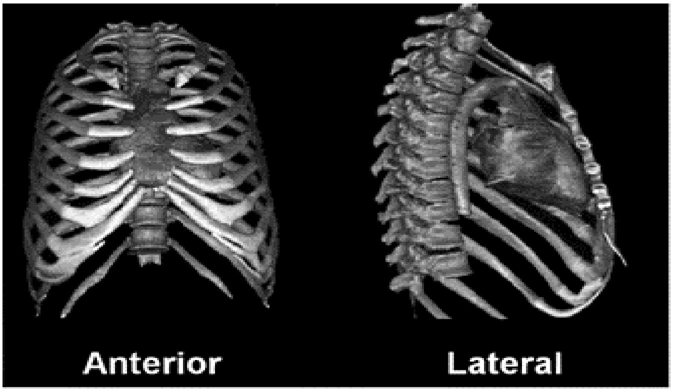

Figure 3.5 Anatomical relationships with the ribs and costal cartilages (anterior view) and the sternum (lateral view). (Images created with the Visible Human Dissector, Touch of Life Technologies, Colorado, USA.)

cava. The anterior position of the right ventricle makes it most vulnerable to cardiac trauma.[34] The cardiac chambers are supported and protected by the fibrous pericardial layer that extends superiorly to cover the proximal portion (3–5 cm) of the ascending aorta and pulmonary artery. There remains only a small portion of the left atrium that is not protected by the pericardial layer at the origin of the four pulmonary veins.

Injuries to the precordium that penetrate the pericardium may cause single or multiple cardiac injuries.[35] Pericardial injury, intracardiac shunts, valvular perforation, and ventricular laceration are well-documented consequences of penetrating chest trauma.[36–39] Knives, missiles, needle injury, and embolic sources typically cause such damage.[40]

Typical entry sites

Penetrating injuries are generally related to wounds to the precordium, but may also be associated with injuries elsewhere in the chest, neck, or upper abdomen.[34] Injuries to the ventral surface of the chest are most common, with consequent damage to the right ventricle. However, subxiphoid needlestick injuries (pericardiocentesis) and the migration of foreign bodies within the cardiovascular system can also cause serious penetrating injuries.[40]

Types of offending objects

Penetrating cardiac injuries are due to a variety of objects, such as knives, bullets, and other missiles. In addition to these external objects, injury can also be caused by rib displacement or the compression of sternal fragments into cardiac structures in close proximity.[41] As well as directly penetrating injuries, objects can enter the heart via the systemic veins.[42–44] In many patients, the retained missile in the heart results in no ill effects.[45,46] However, if a missile is free or partially protruding into a left cardiac chamber, it should be removed, as the embolization of the object to the systemic arterial circulation may have serious consequences.

Gunshot wounds of the heart are often immediately fatal.[10] A 0.22 caliber bullet usually has a relatively small entry path. Larger, more explosive bullets cause directly perforating injuries and remote massive tissue destruction. It is essential that when a bullet entry site is located, exit site damage be located and assessed. Bullet fragments are highly echogenic (like the echogenicity of a prosthetic valve), with distal acoustic shadowing behind the embedded material. Two-dimensional transthoracic echocardiography has gained a role in precisely locating retained bullet fragments within the heart, and in characterizing associated cardiac and vascular injury before and during extraction.[40,47]

Knife wounds are not always immediately fatal, although these injuries are often more serious than blunt-instrument injuries due to the extent of tissue laceration. Shallow knife wounds may only lacerate the pericardium and not penetrate the cardiac chambers. More deeply penetrating injuries, however, will not only enter the pericardium and the cardiac chambers, but may also lacerate the interventricular septum and perforate valve tissue. The hemodynamic consequence of a knife injury will therefore depend on the degree of valvular regurgitation, the size of the ventricular septal defect, and the pericardial effusion that may be produced.

Clinical presentation of penetrating injuries

The prognosis following a penetrating wound depends upon the nature and extent of the injuries, their rapid assessment, and then surgical correction as required. Cardiac decompensation may be difficult to appreciate clinically because of related injuries such as fractured ribs and pulmonary contusions. Transthoracic echocardiography is easily available, but image quality may be limited by lung trauma, pneumothorax and chest-wall injuries. Transesophageal echocardiography may be the procedure of choice if transthoracic imaging proves inconclusive.[39,48]

Tamponade and pericardial trauma

The circumferential covering of the pericardium makes it almost impossible to avoid damage in the setting of a penetrating cardiac injury. Pericardial injuries may result in the collection of pericardial fluid, slowly or rapidly. As the outer pericardial layer is fibrous and rigid, sudden fluid accumulation may cause tamponade.[34,49] Echocardiographic features of cardiac tamponade include right ventricular collapse during diastole and ventricular dysynchrony related to pericardial constraint and ventricular interdependence. Fluctuating filling of the ventricles related to respiration suggests tamponade physiology with hemodynamic compromise (see Chapter 2).

There are three important considerations when examining for tamponade related to trauma. Firstly, patients may initially be stable after trauma. Slow bleeding (often from pericardial laceration) may lead to tamponade developing late. It may therefore be necessary to repeat the echocardiogram over a number of hours, days, or even weeks.[50,51] Secondly, pericardial hematomas after trauma may be loculated and localized (as is often the case after cardiac surgery), causing isolated compression of a single chamber, such as the right atrium (Figure 3.6). Finally, pericardial wounds which open into the pleura may be associated with free bleeding into the pleural space. Such patients will experience signs and symptoms of hemothorax and loss of circulating blood volume with shock.[51]

The management of penetrating wounds to the heart generally consists of immediate thoracotomy and cardiac

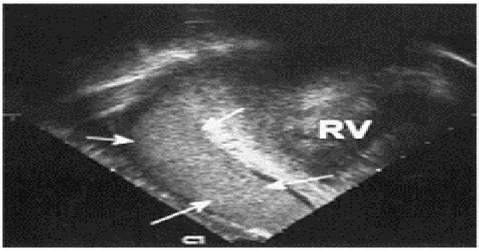

Figure 3.6 Traumatic pericardial hematoma causing compression of the right atrium and right ventricle (RV).

repair.[34,52–54] While arrangements are being made for surgery, the blood volume should be expanded and pericardiocentesis may be performed, to provide time for resuscitation and surgical preparation.[34,49] Echocardiographically guided pericardiocentesis minimizes the risks of pneumothorax and cardiac wall puncture by providing direct visualization of the fluid-filled space and the most direct approach for insertion of the needle-guided catheter.[55,56] Prompt confirmation that the needle is in fact positioned within the fluid (not within the heart) is available from the echocardiogram via agitated saline injection.

Ventricle

Most confrontations are fought head on; as a result, the free wall of the right ventricle is the most frequently wounded chamber.[57] Myocardial laceration and perforations can occur, weakening the wall of the ventricle and contributing to the formation of ventricular aneurysms and ventricular arrhythmias.[58] Ventricular aneurysm formation may cause blood flow stagnation with mural thrombus, which may be detectable by echocardiography. Imaging the apex of the left ventricle from the conventional echocardiographic windows does not always allow full appreciation of apical thrombus. By moving the ultrasound transducer one intercostal space higher than is used for the standard apical four-chamber window, and with steep inferior angulation, all four segments of the left ventricular apex can be well visualized in the 'apical short-axis view'.

Injury to the epicardium, myocardium, and endocardium may cause coronary artery injury and thrombosis with myocardial infarction.[59] Complications of penetrating injuries to the coronary arteries and myocardium are not always immediate. Premorbid ventricular function prior to trauma will influence the degree of hemodynamic compromise related to injury. Hemorrhage into a papillary muscle may cause late necrosis and delayed rupture, again highlighting the importance of careful patient monitoring after penetrating cardiac injury.

Salvage rates are lower in patients with penetrating wounds involving thin-walled structures, such as the atria or the pulmonary artery (43% and 67%, respectively), as they rarely seal off spontaneously. Ventricular perforations are associated with higher survival rates, the contractile properties of the ventricular wall acting to seal the perforation.[38]

Ventricular septa I defect

Rupture of the ventricular septum after penetrating cardiac injury may present late and be difficult to detect clinically.[60] Due to the muscular properties of most of the ventricular septum, acquired ventricular septal defects often seal off spontaneously.[38] Communication between the left and right ventricles is best detected by color Doppler echocardiographic imaging (Figure 3.7). Identifying the site and size of an acquired ventricular septal defect will aid the surgical team in their approach. Penetrating injuries causing ventricular septal defect usually have an entry track through the right ventricular free wall, which should be carefully examined during the echocardiogram. Similarly, damage to the valvular and sub valvular apparatus may be associated injuries and should be carefully excluded by the echocardiogram.

Valve trauma

Penetrating injuries to the heart are frequently complicated by valve leaflet perforation and laceration[37–39,48] Typically, valvular function (stenosis and regurgitation) is assessed by two-dimensional, color Doppler, pulsed-wave Doppler and continuous-wave Doppler echocardiography. Mechanical complications such as papillary muscle laceration, transection of chordae, and perforation of valvular leaflets can usually be imaged by two-dimensional echocardiography.

Valvular dysfunction can present with regurgitation, ranging from mild to severe (see Chapters 1 and 13).[37–39,48] Acute severe regurgitation of the atrioventricular valves

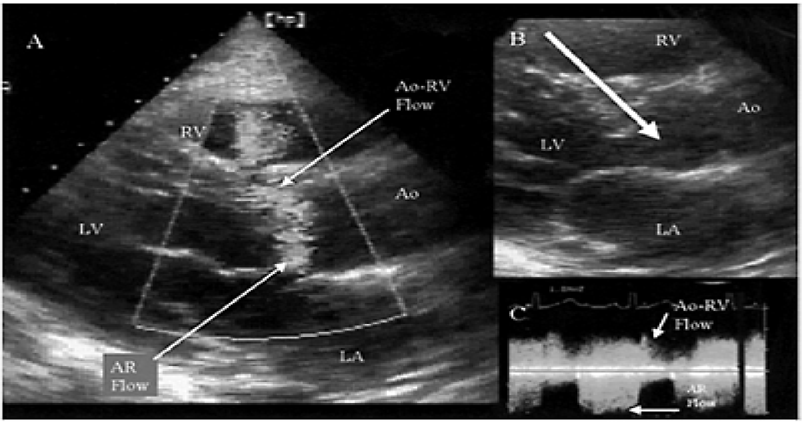

Figure 3.7 Knife stabbing injury. The entry wound through the right ventricle was closed urgently in the emergency room. A continuous machinery murmur was noted. Panel A shows jets of blood from aorta (Ao) to right ventricle (RV), and of aortic regurgitation (AR). Panel B demonstrates clearly the knife track (white arrow) through the base of the ventricular septum, through the right coronary cusp of the aortic valve, and into the aortic root (Ao). Panel C demonstrates continuous Ao-RV continuous-wave Doppler flow above the baseline and diastolic aortic regurgitation flow below the baseline.

usually occurs in the setting of a near normal-sized atrium (left atrial area of ≤20 cm^2, M-mode dimension of ≤40 mm). The consequent rapid increase in volume loading of a small noncompliant chamber can lead to abrupt, large increases in filling pressure with secondary pulmonary edema and/or systemic congestion. This is in contrast to the clinical setting of chronic valvular regurgitation, where the atria enlarge over time and filling pressures are only mildly elevated. Pre-existing valvular disease is not always known prior to injury; therefore, it may not always be possible to appreciate fully a cause-and-effect relationship between trauma and observed valve dysfunction. However, pre-existing valve disease will increase the risk of developing significant valvular disorder after trauma (blunt and penetrating).[61]

Penetrating cardiac trauma may cause minimal valvular regurgitation in the initial phase that may gradually develop into a significant regurgitant lesion requiring surgical intervention.[48] If clinically indicated, repeat transthoracic echocardiograms are useful in assessing the progression of valvular dysfunction after penetrating cardiac injury. Echocardiographic calculations of regurgitant volumes and regurgitant fractions will aid in the quantitation of a progressive valvular lesion.

Great arteries

Penetrating injury to the great vessels should be suspected in any patient in whom a projectile traverses the mediastinum.[62] Presenting signs and symptoms of injury to the great vessels will depend on the size and site of the injury. The pericardium envelopes approximately 3–5 cm of the proximal ascending aorta and pulmonary artery, as they spiral superiorly from the base of the heart. Damage to the great vessels in this area may cause communication between the artery and the pericardial space, frequently resulting in cardiac tamponade.

Penetrating wounds of the great vessels may result in the formation of a false aneurysm with the possibility of subsequent rupture. Echocardiographic features of false aneurysm include the identification of a tear in the intima layer of the vessel and subsequent tearing along the length of the vessel until it comes to the origin of a communicating vessel. Aortocardiac or aortopulmonary communications (fistulae) may complicate a penetrating injury to these vessels with possible resultant congestive cardiac failure.[58]

Iatrogenic injuries

With the rapid development of percutaneous interventions and implantable hardware over the last decade, there has been an inevitable increase in the occurrence of iatrogenic cardiac injuries. The majority of these injuries are of a penetrating nature, as with a pacing wire passing through the right ventricle,[63] a biopsy forceps penetrating through the interventricular septum or valve annulus,[64] an atrial septal puncture needle entering the aorta, or an angioplasty balloon rupturing through a coronary artery.[65] Clearly, echocardiography will focus initially on the detection of pericardial fluid. Indeed, the request for echocardiography as an urgent procedure in the cardiac catheter laboratory

when a patient experiences undue pain or hemodynamic compromise is relatively common. The patients are usually draped and supine, and images are typically first obtained from the subcostal view.

The other iatrogenic cause of cardiac injury is cardiopulmonary resuscitation (CPR). The compressive nature of vigorous CPR causes, rarely, low-velocity blunt trauma to the heart, including valve[66] or ventricular rupture, myocardial contusion, and coronary artery dissection.[67] As discussed in Chapter 4, there is a list of entities that need to be excluded in such patients. It can be difficult to differentiate cardiac abnormalities which are a consequence of CPR from those which were the initial cause of the cardiac arrest.

Conclusions

Thoracic trauma has the potential for rapidly fatal injuries to the heart and great vessels. Many patients do not survive to reach hospital and undergo imaging investigations. In those who do, echocardiography, particularly by the transesophageal route, offers rapidly available, accurate data about the pathoanatomy and allows the treating team to assess stability and plan possible surgical management. The consequences of delays or errors in diagnosis can be immediate and drastic. This is an area of echocardiography which should be undertaken by the most experienced operators where possible. Where doubt remains after echocardiography, particularly in situations with suspected trauma to the ascending aorta, there should be no hesitation for recommending supplementary investigations in these critically ill patients.

References

1. National Center of Health Statistics, US Department of Health and Human Services, Service PH. Monthly Vital Statistics Report, Advance Report of Final Mortality. Statistics 1992; 43:1–76.
2. Pearson GD, Karr SS, Trachiotis GD, Midgley FM, Eichelberger MR, Martin GR. A retrospective review of the role of transesophageal echocardiography in aortic and cardiac trauma in a level I pediatric trauma center. J Am Soc Echocardiogr 1997; 10:946–55.
3. Chirillo F, Totis O, Cavarzerani A, et al. Usefulness of transthoracic and transoesophageal echocardiography in recognition and management of cardiovascular injuries after blunt chest trauma. Heart 1996; 75:301–6.
4. Lancaster GL, DeFrance JH, Borruso JJ. Air-bag-associated rupture of the right atrium. N Engl J Med 1993; 328:358.
5. Sybrandy KC, Cramer MJ, Burgersdijk C. Diagnosing cardiac contusion: old wisdom and new insights. Heart 2003; 89:485–9.
6. Crown LA, Hawkins W. Commotio cordis: clinical implications of blunt cardiac trauma. Am Fam Physician 1997; 55:2467–70.
7. Pontillo D, Capezzato A, Achilli A. Bifascicular block complicating blunt cardiac injury: a case report and review of the literature. Angiology 1994; 45:883–90.
8. Karalis DG, Victor MF, Davis GA, et al. The role of echocardiography in blunt chest trauma: a transthoracic and transesophageal echocardiographic study. J Trauma 1994; 36:53–8.
9. Weiss RL, Brier JA, O’Connor W, Ross S, Brathwaite CM. The usefulness of transesophageal echocardiography in diagnosing cardiac contusions. Chest 1996; 109:73–7.

10. Karo IL, Kushimoto S, Mashiko K. Blunt traumatic rupture of the heart: an experience in Tokyo. J Trauma 1994; 36:859–63.
11. Moront M, Lefrak EA, Akl BF. Traumatic rupture of the interventricular septum and tricuspid valve: case report. J Trauma 1991; 31:134–6.
12. May AK, Patterson MA, Rue LW, Schiller HJ, Rotondo MF, Schwab CW. Combined blunt cardiac and pericardial rupture: review of the literature and report of a new diagnostic algorithm. Am Surg 1999; 65:568–74.
13. Brathwaite CE, Rodriguez A, Turney SZ, Dunham CM, Cowley R. Blunt traumatic cardiac rupture. A 5-year experience. Ann Surg 1990; 212:701–4.
14. Sold M, Silber R, Hopp H, Meesmann M, Ertl G. A successful procedure in mitral valve rupture accompanied by rupture of the papillary muscle and the chordae tendinae following multiple injuries and blunt thoracic trauma. Anaesthesist 1989; 38:262–5.
15. van Son JA, Danielson GK, Schaff HV, Miller FA. Traumatic tricuspid valve insufficiency. Experience in thirteen patients. J Thorac Cardiovasc Surg 1994; 108:893–8.
16. Salehian O, Mulji A. Tricuspid valve disruption and ventricular septal defect secondary to blunt chest trauma. Can J Cardiol 2004; 20:231–2.
17. Sabbah HN, Stein PD, Hawkins ET, Viano DC, Vostal JJ. Extrinsic compression of the coronary arteries following cardiac trauma in dogs. J Trauma 1982; 22:937–43.
18. Gaspard P, Clermont A, Villard J, Amiel M. Non-iatrogenic trauma of the coronary arteries and myocardium: contribution of angiography—report of six cases and literature review. Cardiovasc Intervent Radiol 1983; 6:20–9.
19. Fu M, Wu CJ, Hsieh MJ. Coronary dissection and myocardial infarction following blunt chest trauma. J Formos Med Assoc 1999; 98:136–40.
20. Tun A, Khan IA. Myocardial infarction with normal coronary arteries: the pathologic and clinical perspectives. Angiology 200; 52:299–304.
21. Shapiro MJ, Wittgen C, Flynn MS, Zuckerman DA, Durham RM, Mazuski JE. Right coronary artery occlusion secondary to blunt trauma. Clin Cardiol 1994; 17:157–9.
22. Greendyke RM. Traumatic rupture of aorta; special reference to automobile accidents. JAMA 1966; 195:527–30.
23. Ayella RJ, Hankins JR, Turney SZ, Cowley RA. Ruptured thoracic aorta due to blunt trauma. J Trauma 1977; 17:199–205.
24. Vlahakes GJ, Warren RL. Traumatic rupture of the aorta. N Engl J Med 1995; 332:389–90.
25. Pate JW, Fabian TC, Walker W. Traumatic rupture of the aortic isthmus: an emergency? World J Surg 1995; 19:119–25.
26. Tsoukas A, Stathoulopoulos A, Tsatsoulis P, et al. Aortic transsection after blunt chest trauma. Echocardiography 2001; 18:385–8.
27. Wright-Smith G, Davison MB, Woodford S, McCarthy J, Burstow DJ. The utility of transesophageal echocardiography in the diagnosis of traumatic aortic injury. Aust N Z J Med 1995; 25(Suppl):45.
28. Vignon P, Lagrange P, Boncoeur MP, François B, Gastinne H, Lang RM. Routine transesophageal echocardiography for the diagnosis of aortic disruption in trauma patients without enlarged mediastinum. J Trauma 1996; 40:422–7.
29. Smith MD, Cassidy JM, Souther S, et al. Transesophageal echocardiography in the diagnosis of traumatic rupture of the aorta. N Engl J Med 1995; 332:356–62.
30. Vignon P, Guéret P, Vedrinne JM, et al. Role of transesophageal echocardiography in the diagnosis and management of traumatic aortic disruption. Circulation 1995; 92:2959–68.
31. Roisinblit JM, Allende NG, Neira JA, et al. Local thrombus as an isolated sign of traumatic aortic injury. Echocardiography 2002; 19:63–5.
32. Brooks SW, Young JC, Cmolik B, et al. The use of transesophageal echocardiography in the evaluation of chest trauma. J Trauma 1992; 32:761–5.

33. Vignon P, Rambaud G, François B, Preux PM, Lang RM, Gastinne H. Quantification of traumatic hemomediastinum using transesophageal echocardiography: impact on patient management. Chest 1998; 113:1475–80.
34. Symbas PN, Harlaftis N, Waldo WJ. Penetrating cardiac wounds: a comparison of different therapeutic methods. Ann Surg 1976; 183:377–81.
35. Symbas PN. Cardiothoracic Trauma: Current Problems in Surgery. St Louis, MO: Mosby-Year Book, 1991:742–97.
36. Tesinsky L, Pirk J, al-Hiti H, Malek I. An isolated ventricular septal defect as a consequence of penetrating injury to the heart. Eur J Cardiothorac Surg 1999; 15:221–3.
37. Jenson B, Kessler RM, Follis F, Wernly JA. Repair of atrial septal defect due to penetrating trauma. Tex Heart Inst J 1993; 20: 241–3.
38. Wilson WR, Coyne JT, Greer GE. Mitral regurgitation as a late sequela of penetrating cardiac trauma. J Heart Valve Dis 1997; 6:171–3.
39. Hans Pasteuning W, Wonnink-de Jonge WF, van Berge Henegouwen DP, van der Aa MAC, Penn OM. Acquired ventricular septal defect and mitral insufficiency without pericardial effusion after stab wound to the chest. Am Soc Echocardiogr 1998; 11:483–6.
40. Xie SW, Picard MH. Two-dimensional and color Doppler echocardiographic diagnosis of penetrating missile wounds of the heart: chronic complication from intracardiac course of a bullet. J Am Soc Echocardiogr 1992; 5:81–4.
41. Wilson RF, Bassett JS. Penetrating wounds of the pericardium or its contents. JAMA 1966; 195:513–17.
42. Bland EF, Beebe GW. Missiles in the heart: a 20 year follow-up report of world war cases. N Engl J Med 1966; 274:1039–46.
43. Symbas PN, Hatcher CR Jr, Mansour KA. Projectile embolus of the lung. J Thorac Cardiovasc Surg 1968; 56:97–103.
44. Symbas PN, Harlaftis N. Bullet emboli in the pulmonary and systemic arteries. Ann Surg 1977; 185:318–20.
45. Symbas PN, Picone AL, Hatcher CR Jr, Vlasis SE. Cardiac missiles: a review of the literature and personal experience. Ann Surg 1990; 211:639–48.
46. Symbas PN, Vlasis SE, Picone AL, Hatcher CR Jr. Missiles in the heart. Ann Thorac Surg 1989; 48:192–4.
47. Fry SJ, Picard MH, Tseng JF, Briggs SM, Isselbacher EM. The echocardiographic diagnosis, characterisation and extraction guidance of cardiac foreign bodies. J Am Soc Echocardiogr 2000; 13:232–9.
48. Rywik T, Sitkowski W, Cichocki J, Rajecka A, Suwalski K. Acute mitral regurgitation caused by penetrating chest injury. J Heart Valve Dis 1995; 4:293–5.
49. Cooper FW Jr, Stead EA Jr, Warren JV. The beneficial effect of intravenous infusions in acute cardiac tamponade. Ann Surg 1944; 120:822–5.
50. Raney JL, Kennedy ES. Delayed cardiac tamponade following a stab wound: a case report. J Ark Med Soc 1997; 93:589–91.
51. Boyd TF, Strieder JW. Immediate surgery for traumatic heart disease. J Thorac Cardiovasc Surg 1965; 50:305–12.
52. Attar S, Suter CM, Hankins JR, Sequeira A, McLaughlin JS. Penetrating cardiac injuries. Ann Thorac Surg 1991; 51:711–16.
53. Knott-Craig CJ, Dalton RP, Rossouw GJ, Barnard PM. Penetrating cardiac trauma: management strategy based on 129 surgical emergencies over 2 years. Ann Thorac Surg 1992:53:1006–9.
54. Mitchell ME, Muakkassa FF, Poole GV, Rhodes RS, Griswold JA. Surgical approach of choice for penetrating cardiac wounds. J Trauma 1993; 34:17–20.
55. Kopecky SL, Callahan JA, Tajik AJ, et al. Percutaneous pericardial catheter drainage: report of 42 consecutive patients. Am J Cardiol 1986; 58:633–5.

56. Callahan JA. Two-dimensional echcoardiographically guided pericardiocentesis: experience in 117 consecutive patients. Am J Cardiol. 1985; 55:476–9.
57. Jones EW, Helmsworth J. Penetrating wounds of the heart. Thirty years experience. Arch Surg 1968; 96:671–9.
58. Symbas PN, DiOrio DA, Tyras, DH, Ware RE, Hatcher CR Jr. Penetrating cardiac wounds: significant residual and delayed sequelae. J Thorac Cardiovasc Surg 1973; 66:526–32.
59. Konecke LL, Sptizer S, Mason D, Kasparian H, James PM Jr. Traumatic aneurysm of the left coronary artery. Am J Cardiol 1971; 27:221–3.
60. Asfaw I, Thomas NW, Arfulu A. Interventricular septal defects from penetrating injuries of the heart. A report of 12 cases and review of the literature. J Thorac Cardiovasc Surg 1975; 69:450–7.
61. Parmley LF, Manion WC, Mattingly TW. Nonpenetrating traumatic injury of the heart. Circulation 1958; 18:371–6.
62. Symbas PN, Kourias E, Tyras DH, Hatcher CR Jr. Penetrating wounds of the great vessels. Ann Surg 1974; 179:757–65.
63. Gershon T, Kuruppu J, Olshaker J. Delayed cardiac tamponade after pacemaker insertion. J Emerg Med 2000; 18:355–9.
64. Katta S, Akosah K, Stambler B, Salter D, Guerraty A, Mohanty PK. Atrioventricular fistula: an unusual complication of endomyocar-dial biopsy in a heart transplant recipient. J Am Soc Echocardiogr 1994; 7:405–9.
65. Flynn MS, Aguirre FV, Donohue TJ, Bach RG, Caracciolo EA, Kern MJ. Conservative management of guidewire coronary artery perforation with pericardial effusion during angioplasty for acute inferior myocardial infarction. Cathet Cardiovasc Diagn 1993; 29:285–8.
66. Gerry JL, Bulkley BH, Hutchins GM. Rupture of the papillary muscle of the tricuspid valve. A complication of cardiopulmonary resuscitation and a rare cause of tricuspid insufficiency. Am J Cardiol 1977; 40:825–8.
67. Krischer JP, Fine EG, Davis JH, Nagel EL. Complications of cardiac resuscitation. Chest 1987; 92:287–91.

4
Echocardiography in cardiac arrest

Gregory M Scalia

Key points

- Echocardiography during cardiac arrest, particularly when there is a cardiac rhythm, with or without a palpable pulse or detectable pressure, can provide useful information to help direct the resuscitation team.
- The technical aspects of obtaining images are frequently extremely difficult, and time pressure can be intense.
- Echocardiography operators should be experienced and have the training and equipment available to proceed immediately to transesophageal echocardiography (TEE) if required.

Cardiac arrest situations justifiably provoke a sense of urgency and drama for the attending staff. Seconds matter, as the length of time a patient suffers cardiac arrest clearly has a direct influence on outcome. Many cardiac arrest situations occur suddenly, without warning or obvious precipitant.[1] On discovery of a patient in cardiac arrest, the process of emergency resuscitation begins, with a view to restoring cardiac output and systemic oxygenation, diagnosis of the underlying cause, and correction of any correctable contributing factors. Emergency echocardiography can play a pivotal role in the diagnostic component of the resuscitation process.[2,3]

In the hospital setting, cardiac arrest situations take several forms, which differ somewhat in the requirements for echocardiography. Out-of-hospital cardiac arrests, usually brought in by ambulance and paramedics under full cardiopulmonary resuscitation (CPR), generally have prolonged resuscitation times prior to reaching the arrest team.[4] These patients frequently are severely hypoxic and acidotic, especially if inadequate bystander CPR was undertaken. In these cases, echocardiography is often of limited value because of the very agonal stage of cardiac function at this late stage.

Cardiac arrests which occur in hospital are often (though not always) witnessed and in some cases may be predicted. Indeed, in many situations, echocardiography is called for when a patient is severely hypotensive, prior to full cardiac arrest. It is in these situations where emergency echocardiography is probably of most use. Resuscitation times tend to be shorter, and acidosis has not become so profound, improving the chances of a successful outcome.

Table 4.1 Causes of cardiac arrest and hemodynamic collapse

Electromechanical dissociation and severe hypotension
Left ventricular abnormalities
Severe global left ventricular dysfunction
Regional left ventricular dysfunction
Coronary disease
Sarcoid
Hypertrophic cardiomyopathy
Rare myocardial diseases
Valvular heart disease
Native valves
Prosthetic valves
Pericardial tamponade
Aortic dissection
Pulmonary embolus
Severe hypovolemia/vasodilation
Miscellaneous conditions
Arrhythmic cardiac arrest
Asystole—end-stage cardiac arrest
Ventricular arrhythmia

Therefore, cardiac arrest requiring resuscitation typically is divided into two broad categories:[5] arrhythmic (asystole/ventricular fibrillation), and electromechanical dissociation (EMD), in which there is an organized rhythm (sinus rhythm, ventricular tachycardia, etc.) without a palpable pulse (Table 4.1). For the purpose of this discussion, this latter group should also be considered to include patients with an organized rhythm and severe hypotension (for example, systolic blood pressure of <40 mmHg). It should be noted that many cases that begin with profound hypotension or with frank electromechanical dissociation will progress with time and increasing hypoxia and acidosis to ventricular fibrillation and, of course, if not corrected, to asystole.

Logistical aspects

Cardiac arrest situations typically involve many staff gathered around the patient, including several medical staff, nursing staff, and technicians. The atmosphere is usually hectic, and the situation is often cramped and strained. Typically, there will be an operator at the head ventilating the patient, an operator on one side of the chest undertaking external cardiac compression, and operators at the arms obtaining intravenous access and administering drugs. By the time echocardiography has been called for, the resuscitation has usually been going on for some time.

Transthoracic echocardiography is most commonly utilized in these situations. Typically, the scanner is placed near the patient's bedside, opposite the operator

undertaking external cardiac compression. Because of the supine position of the patient, images are usually best obtained first from the subcostal window, often during a pause in the CPR. Parasternal views may be contributory. Apical views often provide very limited information.

Transesophageal echocardiography (TEE) during resuscitation has the great advantage of increased image clarity and is usually easily performed in the intubated patient.[6] Transesophageal probes are resistant to defibrillation voltages (though the probe should not be touched during the actual shock). TEE is particularly useful if certain tamponade, are suspected. The diagnostic accuracy of TEE specific entities, such as dissection and isolated atrial during resuscitation compared with surgical or autopsy data has been shown to be high.[7]

Medicolegal considerations are also relevant to echocardiography during resuscitation. The videotape (or digital record) of the echo represents a time-coded recording of the resuscitation process and the interventions that may be undertaken (such as pericardiocentesis and defibrillation). The echocardiography report should preferably include a chronology of the events for future reference. Furthermore, cardiac arrest may be the result of some misadventure, accident, or violent trauma; thus, the data from the echocardiography may become part of subsequent investigations and review.

Electromechanical dissociation and severe hypotension

Echocardiography is often called for during the resuscitation process in patients with electromechanical dissociation (EMD) or severe hypotension. At this stage, before severe hypoxia and acidosis have supervened, correct diagnosis of the underlying etiology of the EMD/hypotension may lead the situation toward remedy. Therefore, the echocardiography operator should systematically consider and assess for each of the potential causes listed below.

Left ventricular abnormalities

Severe global left ventricular dysfunction

Left ventricular events are commonly the precipitant of cardiac arrest. Echocardiography during resuscitation in these patients initially assesses overall left ventricular function. Severe global dysfunction is commonly seen after prolonged resuscitation regardless of the inciting disorder, and may therefore be relatively nonspecific. However, finding severe global dysfunction immediately on arrest or in patients with severe hypotension suggests a diffuse cardiomyopathic process such as idiopathic/familial cardiomyopathy,[8] ischemic cardiomyopathy, alcoholic cardiomyopathy, peripartum cardiomyopathy,[9] etc. Right ventricular dysfunction is a variable finding in such cases. The finding on echocardiography of severe global left ventricular dysfunction should direct the resuscitation process toward inotrope administration.

Regional left ventricular dysfunction

Coronary disease is the most common causes of sudden cardiac death.[10] Cardiac arrest in patients with ischemic heart disease can occur as a consequence of previous infarct with scarring and secondary arrhythmia, or as a result of an acute ischemic event with a region of myocardium generating arrhythmia (Figure 4.1). Echocardiography during resuscitation in these patients is directed toward the detection of specific regional wall motion abnormalities. It is sometimes possible to infer

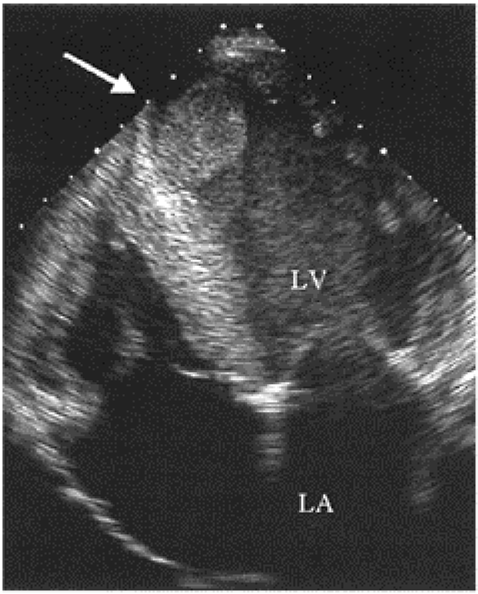

Figure 4.1 Large anteroapical infarct with thrombus (arrow) as a cause of cardiac arrest. LA: left atrium; LV: left ventricle.

that a given wall motion abnormality is old if the area is thinned or calcified. An attempt should also be made to identify the coronary distribution involved (left main coronary, left anterior descending, or circumflex and right coronary artery) (see Chapter 14). The finding of a regional wall motion abnormality during resuscitation may direct efforts toward anti-ischemic medical therapies and possible revascularization procedures.[11]

Echocardiography in cardiac arrest is particularly useful for diagnosing the acute catastrophic mechanical complications of myocardial ischemia. Ischemic papillary muscle rupture is typically associated with fulminating cardiogenic shock progressing to profound hypotension and finally EMD. This lesion causes acute severe mitral regurgitation into usually a small noncompliant left atrium with associated severe

pulmonary venous hypertension and pulmonary edema. Echocardiography demonstrates complete disruption of the mitral apparatus at its connection into the papillary muscle head.[12] Often the leaflets flail back into the left atrium with a fragment of muscle visible on the ends of the chords. The posteromedial papillary muscle is most frequently involved secondary to

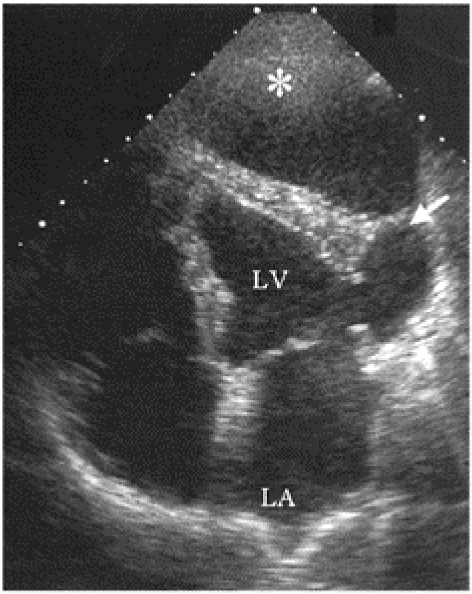

Figure 4.2 Left ventricular-contained free-wall rupture (arrow) with large pseudoaneurysm (*) causing severe hypotension. LA: left atrium; LV: left ventricle.

the distal right coronary artery or circumflex ischemia.[13] Of note, it is common not to perceive a regional wall motion abnormality in these cases, presumably secondary to the massive unloading of the ventricle by the mitral regurgitation (see Chapters 13 and 15).

Ventricular rupture occurs as a consequence of acute myocardial infarction and has become a familiar phenomenon since the large-scale introduction of thrombolysis (see Chapter 15). Perhaps intramural hemorrhage into necrotic myocardial segments causes tissue disruption and subsequent rupture. Notwithstanding the mechanism, ventricular rupture can occur through the free wall into the pericardium[14] (causing tamponade—see below) or contained rupture with pseudoaneurysm (Figure 4.2). Rupture can also occur through the interventricular septum, causing ventricular septal defect formation, which, if large, can also be associated with cardiovascular collapse.[15,16]

Echocardiography can also detect rarer causes of regional left ventricular dysfunction in patients suffering cardiac arrest. Patients with endocarditis, particularly of the aortic

valve, have been observed to suffer acute ischemic events secondary to coronary embolus. Patients

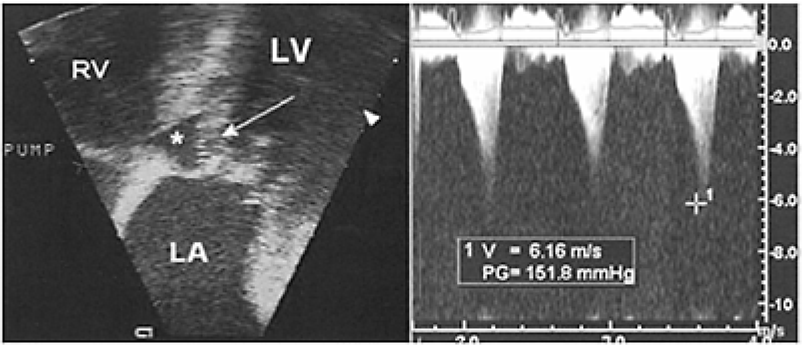

Figure 4.3 Severe hypertrophic cardiomyopathy with systolic anterior motion (SAM) of the mitral valve (arrow) into the left ventricular outflow tract (*). Continuous-wave Doppler (right panel) shows severe outflow tract obstruction, with a calculated peak gradient of 151 mmHg. Note that the late peaking is 'dagger-shaped'. RV: right ventricle; LV: left ventricle; LA: left atrium.

with cardiac sarcoid are particularly prone to ventricular arrhythmias and thence cardiac arrest. In such patients, regional wall motion abnormalities which do not conform to recognized coronary distributions are often seen. These areas are often thinned sometimes with nodular fibrotic lesions within the myocardium.[17]

Hypertrophic cardiomyopathy

Patients with hypertrophic cardiomyopathy are prone to sudden cardiac death from several mechanisms.[18] Primary ventricular arrhythmias are well documented, possibly secondary to the myofibrillar disarray typical of this condition.[19] Profound vasodepressor episodes mediated by baroreceptor reflexes have also been implicated as causal in the sudden death of these patients. Finally, in situations where volume underloading occurs (such as during noncardiac surgery, with prolonged fasting and vasodilator anesthetic agents) patients with hypertrophic cardiomyopathy may develop severe outflow tract obstruction secondary to systolic anterior motion of the mitral valve, which itself is complicated by severe mitral regurgitation (Figure 4.3). These patients, who typically are elderly and have the proximal septal hypertrophy form of the disease,[20] can therefore

convert from their normal, relatively stable functional anatomy to a ventricular configuration where there is both severe outflow tract obstruction and severe mitral regurgitation in a very short period of time. Such findings during echocardiography at the time of resuscitation may prompt the administration of volume-loading and negative inotropes such as beta blockers.

Rare myocardial causes of cardiac arrest

Infiltrative conditions of the myocardium, such as amyloid, are associated with sudden cardiac death secondary to pump failure and arrhythmia.[21] Typically, end-stage amyloid (and other infiltrative conditions) will be manifest by generalized thickening of the myocardium of both ventricles and atria and of valvular tissue. Echocardiography should determine whether the condition is associated with preserved or diminished systolic function.

Valvular heart disease

Critical valvular disease has become progressively less common in Western communities with improved levels of medical care and surgical intervention. Notwithstanding this, end-stage valve disease, whether undiagnosed or intentionally untreated because of age or other comorbidities, can precipitate cardiac arrest. Critical aortic stenosis, easily detected by echocardiography during resuscitation, is a well-recognized cause of sudden cardiac death.[22,23] Low cardiac output, compensatory left ventricular hypertrophy, and relative (or actual) myocardial ischemia can lead to malignant arrhythmia and arrest.[24] Critical mitral stenosis rarely presents with cardiac arrest, rather manifesting with progressive congestive heart failure. Rare cases with large mobile left atrial thrombus causing 'ball-valve' obstruction of the mitral orifice have been seen to cause sudden death.

Regurgitant lesions are usually slowly progressive and usually manifest in most patients as progressive congestive heart failure. Cardiac arrest can occur when there is acute fulminant regurgitation, particularly of the mitral valve.[25] This can occur as a result of ischemic heart disease (papillary muscle rupture—see above), myxomatous chordal rupture, and trauma (see Chapters 3, 13 and 15). Mitral valve prolapse has been associated with sudden cardiac death by unclear mechanisms even in the absence of recent major increases in regurgitant volume.[26] Endocarditis can be associated with cardiac arrest by either direct valvular destruction causing acute severe regurgitation or large vegetations causing ball-valve obstruction of either the valve itself or a coronary ostia, and by coronary embolism.[27]

Failure of prosthetic heart valves, particularly mechanical valves, can be abrupt and associated with massive hemodynamic collapse and subsequent cardiac arrest.[28] Leaflet motion can be impaired by thrombus (particularly when anticoagulation has become sub therapeutic), vegetation, or pannus.[29] The atrioventricular valves seem more vulnerable than the pulmonary and aortic valve prostheses. Echocardiographic examination should carefully delineate the leaflet range of motion and transvalvular gradients. TEE will usually be required because of acoustic shadowing by the valve material (see Chapter 17).

The finding of critical valvular heart disease on echocardiography during resuscitation should prompt the team to consider urgent surgical consultation,[30] after restoration of the circulation with inotropes, fluids, and perhaps intra-aortic balloon pump insertion. There may of course be other mitigating factors in such decisions.

Pericardial tamponade

In the case of out-of-hospital cardiac arrest, pericardial disease is a relatively rare cause of cardiac arrest. In severe cases, viral pericarditis can lead to massive pericardial effusion with hemodynamic collapse.[31] Connective tissue disease,[32] hypothyroidism,[33] and malignancy[34] can also cause large effusions,[35] but because of the relatively slow accumulation of fluid, these generally become clinically evident before they reach the stage of hemodynamic collapse or cardiac arrest.

Within the hospital setting, particularly in hospitals with cardiac surgical services, cardiac tamponade represents a very common cause of hemodynamic collapse and cardiac arrest (see Chapter 2).[36] Bleeding into the pericardium is not uncommon, particularly in the current era of early cardiac surgery in acute coronary syndromes where various antiplatelet and antithrombotic medications are administered.

When called to the resuscitation of a postcardiac surgical patient, the echocardiography operator needs to be aware of the various configurations of pericardial fluid in these patients.[37] Pericardial effusions may be liquid and appear black on the echocardiography screen (Figure 4.4). This liquid can be frank blood, serous fluid, or a mixture (serosanguineous, or ‘pink’, fluid). In these cases, the fluid is often circumferential and may be visualized from the parasternal, apical, and subcostal views. Less frequently, truly liquid effusions may be loculated.

More frequently, pericardial effusions after cardiac surgery are semisolid or have appearances consistent with hematoma (see Figure 3.6). Large fibrin strands may be seen within the material. In these cases, it is very common for the material to be loculated over one portion of the heart, especially the atria. Transthoracic imaging during resuscitation (or at any other time) often proves inadequate in examining for loculated pericardial effusion. Limited image resolution in the far-field and difficulty in differentiating pericardial material from surrounding structures are common problems. The assessment of pericardial tamponade as a cause of hemodynamic collapse/cardiac arrest will often therefore require TEE.

Large pericardial effusions and loculated collections over the atria cause hemodynamic compromise and eventual cardiac arrest by their space-occupying effects and ensuing compression of the heart, which causes impaired diastolic filling. In the patient with some cardiac output, the echocardiography operator should try to assess the Doppler findings of tamponade by interrogating transmitral flow, transtricuspid flow, and pulmonary and hepatic vein flows.[38] However, in the more drastic cases, where there is complete hemodynamic collapse (and perhaps CPR in progress), these subtle findings may be extremely difficult to demonstrate.

However, it is in these very situations that echocardiography operators are perhaps under more immediate pressure than at any other time in their clinical practice, as an immediate life-saving decision has to be made as to whether urgently to reopen the chest.

Therefore, the operator should look for other signs such as chamber compression or inversion, paradoxical septal motion, and ventricular underfilling.

Once the diagnosis of tamponade is made, the echocardiography operator will often be asked whether the effusion can be drained percutaneously, or whether emergency reopening is required.[39] In general, large liquid effusions can be drained percutaneously, whereas smaller effusions or those that are semisolid or loculated require

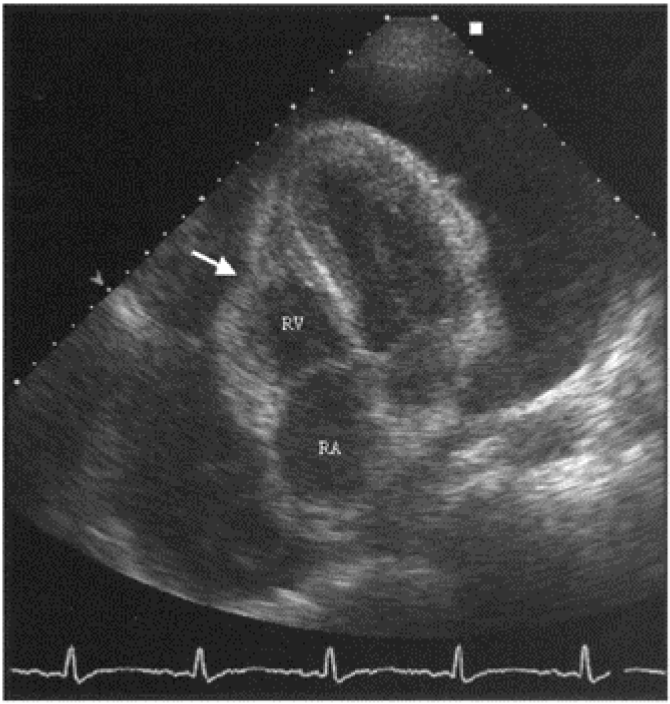

Figure 4.4 Large circumferential liquid pericardial effusion causing tamponade with compression of the right ventricular (RV) free wall (arrow). RA: right atrium.

open evacuation. The approach used for percutaneous pericardiocentesis will often be guided by the echocardiogram (see Chapter 2). The largest diameter of ‘free fluid’ should be sought where the drainage needle is less likely to accidentally strike the heart. Entry sites can be obtained from the parasternal (right or left), apical, or, most commonly, the subcostal position.[40,41]

Once the entry site is decided upon, the echocardiography operator demonstrates the angle of attack and the depth required for the drainage procedure. Typically, the entry site is then prepared and draped, and the drainage process begun. It is common for the echocardiography operator to observe the diminution of the fluid volume from a second

acoustic window (such as the apical window if the fluid is being drained from the subcostal approach). In this way, the team can be sure that drainage is complete before the pericardiocentesis catheter is withdrawn. Probe-mounted needle guides have also been used to provide continuous direct vision during insertion.[42] The utility of this technique during the hustle of a cardiac arrest remains to be demonstrated.

Pericardiocentesis during cardiopulmonary resuscitation is often a very difficult procedure and is usually undertaken during a pause in external cardiac compressions. It is not uncommon for dark red fluid to be aspirated up the guide needle. All present will then simultaneously wonder whether the needle has entered a hemopericardium or the right ventricle. The echocardiography operator can answer this question quickly with the injection down the needle of a few milliliters of saline[43] (or echo contrast agent[44]). Microbubbles will then be seen in one or other of these spaces, and appropriate action may be taken.

Aortic dissection

Aortic dissection can occur naturally as a result of hypertension and medial necrosis of the aorta (see Chapter 12). In the postcardiac surgical situation, aortic dissection can occur as an iatrogenic consequence of instrumentation and cannulation of the ascending aorta. The life-threatening

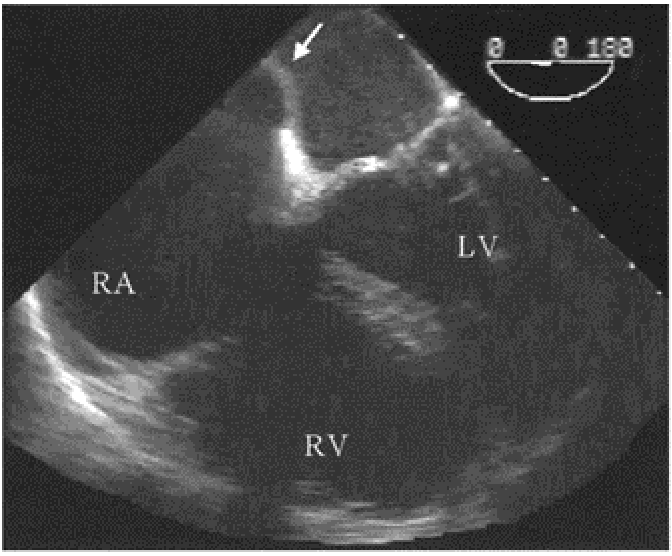

Figure 4.5 Transesophageal, four-chamber view of a patient suffering cardiac arrest secondary to massive pulmonary embolism. Note the dilated right atrium (RA) and right ventricle

(RV), which was severely hypokinetic Right atrial pressure is clearly raised above left atrial pressure, as evidenced by the bowing of the atrial septum from right to left (white arrow). LV: left ventricle.

complications of aortic dissection relate to proximal extension into the pericardium, which causes massive hemopericardium and tamponade, and dissection of the coronary ostia, which causes myocardial ischemia.[45] The transition from stable proximal aortic dissection to massive hemopericardium with tamponade and subsequent death is unpredictable in its timing and catastrophic.

In patients undergoing resuscitation, particularly after cardiac surgery, assessment of the proximal aorta should be made for evidence of a dissection flap. This finding is not easily detected with transthoracic echocardiography. Supportive evidence might include the finding of a dilated ascending aorta, aortic regurgitation, and/or evidence of a dissection flap in the arch (suprasternal view) or abdominal aorta. If the suspicion of aortic dissection is high in a patient with cardiac arrest, transesophageal echo will most likely be required.

Pulmonary embolus

Patients who collapse suddenly with electromechanical dissociation, particularly in the hospital setting where there may have been a period of immobilization, or after prolonged plane travel, are highly likely to have suffered massive pulmonary embolism (see Chapter 8).[46] Complete or near-complete obstruction of the pulmonary arterial system by embolic material from the legs or great veins of the pelvis causes acute and catastrophic right heart failure with profound decreases in cardiac output.[47,48]

In patients who have not suffered pulmonary arterial hypertension prior to the event, the right ventricle will respond to this abrupt pressure overload with immediate dilation and severe dysfunction. Pulmonary arterial systolic pressure (or right ventricular systolic pressure) may be elevated, but is often not markedly so. Indeed, it is most uncommon for such a patient to achieve pulmonary arterial systolic pressures of >60 mmHg. In patients where there has been previous pulmonary hypertension (as in chronic obstructive airways disease or chronic recurrent pulmonary emboli), the right ventricle will have hypertrophied with time. The hypertrophied right ventricle will react to the acute potentially fatal pulmonary embolus by increasing in contractility and generating pressures of 90 mmHg or more.

Echocardiography during the resuscitation of patients with massive pulmonary embolism[49] may therefore demonstrate a dilated and hypokinetic right ventricle (Figure 4.5). Secondarily, the left ventricle will be under-filled and often quite hyperdynamic (in an attempt to prop up cardiac output).[50] Right atrial pressure will be greater than left atrial pressure, and thus the interatrial septum will be seen to bow from right to left.[51]

Actual embolic material may be seen in the main pulmonary artery.[52] This is often best appreciated with TEE at an approximately 70–90° plane. Thrombus in-transit can

sometimes be seen in the right atrium or ventricle[53] and is occasionally observed to be straddling a patent foramen ovale. Failure to visualize embolic material with echocardiography does *not* exclude the diagnosis and indeed is not uncommon even in confirmed cases of pulmonary embolus. The presumptive diagnosis of pulmonary embolus made by echocardiography during resuscitation may prompt the team to consider emergency administration of anticoagulants and thrombolytics, or to undertake embolectomy procedures—either percutaneous or open surgical.

Severe hypovolemia

Cardiac arrest ‘code’ calls are often made for patients found collapsed and pulseless for reasons unknown. Emergency echocardiography is frequently called for, and the images demonstrate no specific abnormality. The left ventricle is small and vigorous in function. In some cases, there is cavity obliteration. The right ventricle is also small and dynamic. There is no pericardial fluid and usually no significant valvular abnormality. These findings are typical of acute severe hypovolemia and shock. This can be seen in patients suffering profound hemorrhage, massive fluid losses into interstitial spaces as in the postoperative state, and in patients suffering septic shock. These findings during resuscitation should prompt the team to undertake aggressive fluid resuscitation and vasopressor therapy.

Rare causes of cardiac arrest detected by echocardiography

Cardiac tumors can lead to ball-valve obstruction causing hemodynamic collapse. This is particularly true of pedunculated atrial masses, such as myxomas,[54] which prolapse through the mitral valve orifice, causing obstruction (see Chapter 16). Air embolism secondary to intravenous infusions, or intraperitoneal insufflation for laparoscopy may be detected by echocardiography as air bubbles in the right atrium and ventricle.[55] Cardiac dysfunction after intracranial hemorrhage or trauma may cause arrest. Echocardiography may reveal regional left ventricular dysfunction, often in a non-coronary distribution.[56] Often this will involve the mid- and distal ventricle with the basal portion of all segments contracting well.

Arrhythmogenic right ventricular dysplasia is a hereditary muscular abnormality of the right ventricular free wall with fatty infiltrates and wall motion abnormalities and thinning. Although magnetic resonance imaging (MRI) is currently the standard for diagnosis, echocardiography during resuscitation of patients with a family history of this disease should look for typical features. Similarly, patients with a personal or family history of Friedreich’s ataxia or other neuromuscular diseases[57] should be carefully screened for the echocardiographic features of these diseases during resuscitation.

Echocardiography in asystolic (end-stage) cardiac arrest

When resuscitation time has been long and the underlying rhythm has become asystolic (flat-line), echocardiography during cardiac arrest has relatively limited contributions to make.[58] Many of the features listed above will no longer be present, as there is no cardiac

rhythm or contractility remaining.[59] Indeed, mechanical cardiac standstill by echocardiography has been associated with a uniformly poor outcome in some series.[60] Of course, pericardial fluid will persist. However, at this late stage, drainage will often have little benefit. Notwithstanding this, vigorous resuscitation and administration of epinephrine and other agents may restore rhythm and then allow a more meaningful echocardiographic examination. The presence of cardiac activity late in the resuscitation process has been shown to predict a favorable outcome.[61] Typically, the echocardiography operator will remain with the team until it is clear that no functional rhythm can be achieved.

Training and competency issues

Echocardiography during CPR is often pressured, and frequently the technical aspects of obtaining the images are extremely difficult. The area is crowded, and the time pressure to obtain the data is obvious. Furthermore, life and death decisions need to be made on the data immediately, usually without the time for the operator to consult with other staff. With these issues in mind, it is obvious that operators should have significant experience with routine echocardiography. Moreover, given the limitations of transthoracic echocardiography in detecting and clearly delineating some of the entities listed above (such as dissection and loculated pericardial hematoma), the echocardiography operator should ideally have the training and equipment available to proceed immediately to TEE if required.

Summary

Echocardiography during cardiac arrest, particularly in those cases where there is a cardiac rhythm, with or without a palpable pulse or detectable pressure, can provide useful diagnostic data to help direct the resuscitation team. Imaging is often difficult and time pressure can be intense. However, experienced echocardiography operators can systematically work through the diagnostic possibilities, allowing recommendation of therapeutic interventions which may assist the resuscitation process.

References

1. Kuisma M, Alaspää A. Out-of-hospital cardiac arrests of non-cardiac origin. Epidemiology and outcome. Eur Heart J 1997; 18:1122–8.
2. Varriale P, Maldonado JM. Echocardiographic observations during in-hospital cardiopulmonary resuscitation. Crit Care Med 1997; 25:1717–20.
3. Mullany D, Scalia GM. Use of echocardiography in the diagnosis and management of circulatory shock and cardiac arrest in adults. Contemp Crit Care 2004; 8; 1–6.
4. De Maio VJ, Stiell IG, Wells GA, Spaite DW. Cardiac arrest witnessed by emergency medical services personnel: descriptive epidemiology, prodromal symptoms, and predictors of survival. OPALS study group. Ann Emerg Med 2000; 35:138–46.

5. Andréasson AC, Herlitz J, Bång A, et al. Characteristics and outcome among patients with a suspected in-hospital cardiac arrest. Resuscitation 1988; 39:23–31.
6. Sohn DW, Shin GJ, Oh JK, et al. Role of transesophageal echocardiography in hemodynamically unstable patients. Mayo Clin Proc 1995; 70:925–31.
7. van der Wouw PA, Koster RW, Delemarre BJ, de Vos R, Lampe-Schoenmaeckers AJ, Lie KI. Diagnostic accuracy of transesophageal echocardiography during cardiopulmonary resuscitation. J Am Coll Cardiol 1997; 30:780–3.
8. Corrado D, Nava A, Buja G, et al. Familial cardiomyopathy underlies syndrome of right bundle branch block, ST segment elevation and sudden death. J Am Coll Cardiol 1996; 27:443–8.
9. Lasinska-Kowara M, Dudziak M, Suchorzewska J. Two cases of postpartum cardiomyopathy initially misdiagnosed for pulmonary embolism. Can J Anaesth 2001; 48:773–7.
10. Kannel WB, Gagnon DR, Cupples LA. Epidemiology of sudden coronary death: population at risk. Cardiology 1990; 6:439–44.
11. Borger van der Burg AE, Bax JJ, Boersma E, et al. Impact of percutaneous coronary intervention or coronary artery bypass grafting on outcome after nonfatal cardiac arrest outside the hospital. Am J Cardiol 2003; 91:785–9.
12. Moursi MH, Bhatnagar SK, Vilacosta I, San Roman JA, Espinal MA, Nanda NC. Transesophageal echocardiographic assessment of papillary muscle rupture. Circulation 1996; 94:1003–9.
13. Christ G, Siostrzonek P, Maurer G, Baumgartner H. Partial papillary muscle rupture complicating acute myocardial infarction. Diagnosis by multiplane transoesophageal echocardiography. Eur Heart J 1995; 16:1736–8.
14. Oliva PB, Hammill SC, Edwards WD. Cardiac rupture, a clinically predictable complication of acute myocardial infarction: report of 70 cases with clinicopathologic correlations. J Am Coll Cardiol 1993; 22:720–6.
15. Smyllie J, Dawkins K, Conway N, Sutherland GR. Diagnosis of ventricular septal rupture after myocardial infarction: value of colour flow mapping. Br Heart J 1989; 62:260–7.
16. Obarski TP, Rogers PJ, Debaets DL, Murcko LG, Jennings MR. Assessment of postinfarction ventricular septal ruptures by transesophageal Doppler echocardiography. J Am Soc Echocardiogr 1995; 8:728–34.
17. Nielsen-Kudsk JE. Ventricular fibrillation and cardiac aneurysms caused by sarcoidosis. Ugeskr Laeger 1993; 155:3299–301.
18. Cecchi F, Maron BJ, Epstein SE. Long-term outcome of patients with hypertrophic cardiomyopathy successfully resuscitated after cardiac arrest. J Am Coll Cardiol 1989; 13:1283–8.
19. Spirito P, Maron BJ. Relation between extent of left ventricular hypertrophy and occurrence of sudden cardiac death in hypertrophic cardiomyopathy. J Am Coll Cardiol 1990; 15:1521–6.
20. Dunn FG, Pringle SD. Sudden cardiac death, ventricular arrhythmias and hypertensive left ventricular hypertrophy. J Hypertens 1993; 11:1003–10.
21. Petersen EC, Engel JA, Radio SJ, Canfield TM, McManus BM. The clinical problem of occult cardiac amyloidosis. Forensic implications. Am J Forensic Med Pathol 1992; 13:225–9.
22. Pavlides GS, Cieszkowski J, Timmis GC, O'Neill W. Successful resuscitation of a patient with critical aortic stenosis and cardiac arrest by peripheral cardiopulmonary support system. Cathet Cardiovasc Diagn 1990; 20:120–2.
23. Sorgato A, Faggiano P, Aurigemma GP, Rusconi C, Gaasch WH. Ventricular arrhythmias in adult aortic stenosis: prevalence, mechanisms, and clinical relevance. Chest 1998; 113:482–91.
24. Pellikka PA, Nishimura RA, Bailey KR, Tajik AJ. The natural history of adults with asymptomatic, hemodynamically significant aortic stenosis. J Am Coll Cardiol 1990; 15:1012–17.
25. Ferguson DW, Kiefaber RW, Ziegelman DS, Uphold RE, Jackson RS, Tabakin BS. Acute rupture of myxomatous mitral valve presenting as refractory cardiopulmonary arrest. J Am Coll Cardiol 1987; 9:215–20.

26. Boudoulas H, Schaal SF, Stang JM, Fontana ME, Kolibash AJ, Wooley CF. Mitral valve prolapse: cardiac arrest with long-term survival. Int J Cardiol 1990; 26:37–44.
27. Basmadjian AJ, Ducharme A, Ugolini P, Petitclerc R, Leung TK, Tardif JC. Obstruction of left ventricular outflow tract by vegetation and periaortic abscess. J Am Soc Echocardiogr 2000; 13:869–72.
28. Clark MA. Mechanical failure of a mitral valve prosthesis: an unusual case of sudden unexpected death. J Forensic Sci 1985; 30:552–5.
29. Keeble W, Cobbe SM. Pressure damping, a ‘billowing’ septum, and an eerie silence: perioperative, intermittent obstruction of a mitral valve prosthesis. Heart 2000; 84:E6.
30. Matsuhisa H, Mukouhara N, Obo H, Nakagiri K, Kozawa S, Shida T. Successful emergency aortic valve replacement following percutaneous cardiopulmonary support for the patient with aortic stenosis and cardiac arrest. Jpn J Thorac Cardiovasc Surg 2003; 51:381–3.
31. Permanyer-Miralda G, Sagristá-Sauleda J, Soler-Soler J. Primary acute pericardial disease: a prospective series of 231 consecutive patients. Am J Cardiol 1985; 56:623–30.
32. Kahl LE. The spectrum of pericardial tamponade in systemic lupus erythematosus. Report of ten patients. Arthritis Rheum 1992; 35:1343–9.
33. Gupta R, Munyak J, Haydock T, Gernsheimer J. Hypothyroidism presenting as acute cardiac tamponade with viral pericarditis. J Emerg Med 1999; 17:176–8.
34. Forslund T, Forsen KO, Maenpaa J. Cardiac tamponade due to ovarian carcinoma. Arch Gynecol Obstet 1991; 248:161–5.
35. Keefe DL. Cardiovascular emergencies in the cancer patient. Semin Oncol 2000; 27:244–55.
36. Bommer WJ, Follette D, Pollock M, Arena F, Bognar M, Berkoff H. Tamponade in patients undergoing cardiac surgery: a clinical-echocardiographic diagnosis. Am Heart J 1995; 130:1216–23.
37. Anthi A, Tzelepis GE, Alivizatos P, Michalis A, Palatianos GM, Geroulanos S. Unexpected cardiac arrest after cardiac surgery: incidence, predisposing causes and outcome of open chest cardiopulmonary resuscitation. Chest 1998; 113:15–19.
38. Tsang TS, Oh JK, Seward JB, Tajik AJ. Diagnostic value of echocardiography in cardiac tamponade. Herz 2000; 25:734–40.
39. Wall TC, Campbell PT, O’Connor CM, et al. Diagnosis and management (by subxiphoid pericardiotomy) of large pericardial effusions causing cardiac tamponade. Am J Cardiol 1992; 69:1075–8.
40. Salem K, Mulji A, Lonn E. Echocardiographically guided pericardiocentesis—the gold standard for the management of pericardial effusion and cardiac tamponade. Can J Cardiol 1999; 15:1251–5.
41. Tsang TS, El-Najdawi EK, Seward JB, Hagler DJ, Freeman WK, O’Leary PW. Percutaneous echocardiographically guided pericardiocentesis in pediatric patients: evaluation of safety and efficacy. J Am Soc Echocardiogr 1998; 11:1072–7.
42. Maggiolini S, Bozzano A, Russo P, et al. Echocardiography-guided pericardiocentesis with probe-mounted needle: report of 53 cases. J Am Soc Echocardiogr 2001; 14:821–4.
43. Chiang HT, Lin M. Pericardiocentesis guided by two-dimensional contrast echocardiography. Echocardiography 1993; 10:465–9.
44. Caspari G, Bartel T, Möhlenkamp S, et al. Contrast medium echocardiography-assisted pericardial drainage. Herz 2000; 25:755–60.
45. Garcia-Jimenez A, Peraza Torres A, Martinez Lopez G, Alvarez Dieguez I, Botana Alba CM. Cardiac tamponade by aortic dissection in a hospital without cardiothoracic surgery. Chest 1993; 104:290–1.
46. Early intervention in massive pulmonary embolism. A guide to diagnosis and triage for the critical first hour. Postgrad Med 2002; 111:27–8, 33–4, 39–40 passim.
47. Comess KA, DeRook FA, Russell ML, Tognazzi-Evans TA, Beach KW. The incidence of pulmonary embolism in unexplained sudden cardiac arrest with pulseless electrical activity. Am J Med 2000; 109:351–6.

48. Kurkciyan I, Meron G, Sterz F, et al. Pulmonary embolism as a cause of cardiac arrest: presentation and outcome. Arch Intern Med 2000; 160:1529–35.
49. MacCarthy P, Worrall A, McCarthy G, Davies J. The use of transthoracic echocardiography to guide thrombolytic therapy during cardiac arrest due to massive pulmonary embolism. Emerg Med J 2002; 19:178–9.
50. Rudoni RR, Jackson RE, Godfrey GW, Bonfiglio AX, Hussey ME, Hauser AM. Use of two-dimensional echocardiography for the diagnosis of pulmonary embolus. J Emerg Med 1998; 16:5–8.
51. Jackson RE, Rudoni RR, Hauser AM, Pascual RG, Hussey ME. Prospective evaluation of two-dimensional transthoracic echocardiography in emergency department patients with suspected pulmonary embolism. Acad Emerg Med 2000; 7:994–8.
52. Mabee SW, Mabee CL, Pacht ER. Normal arterial blood gas in a patient with saddle pulmonary artery embolus: diagnosis by transesophageal echocardiography. J Natl Med Assoc 1995; 87:717–19.
53. Hunter JJ, Johnson KR, Karagianes TG, Dittrich HC. Detection of massive pulmonary embolus-in-transit by transesophageal echocardiography. Chest 1991; 100:1210–14.
54. Asai Y, Ichimura K, Kaneko M, Abe T. Treatment of life-threatening huge atrial myxoma: report of two cases. Surg Today 1999; 29:813–16.
55. Sigman DB, Hasnain JU, Del Pizzo JJ, Sklar GN. Real-time transesophageal echocardiography for intraoperative surveillance of patients with renal cell carcinoma and vena caval extension undergoing radical nephrectomy. J Urol 1999; 161:36–8.
56. Kono T, Morita H, Kuroiwa T, Onaka H, Takatsuka H, Fujiwara A. Left ventricular wall motion abnormalities in patients with subarachnoid hemorrhage: neurogenic stunned myocardium. J Am Coll Cardiol 1994; 24:636–40.
57. Moorman JR, Coleman RE, Packer DL, et al. Cardiac involvement in myotonic muscular dystrophy. Medicine 1985; 64:371–87.
58. Blaivas M, Fox JC. Outcome in cardiac arrest patients found to have cardiac standstill on the bedside emergency department echocardiogram. Acad Emerg Med 2001; 8:616–21.
59. Wang FS, Lien WP, Fong TE. Terminal echocardiographic findings during death process in man and dogs. J Formos Med Assoc 1991; 90:31–6.
60. Blaivas M, Fox JC. Outcome in cardiac arrest patients found to have cardiac standstill on the bedside emergency department echocardiogram. Acad Emerg Med 2001; 8:616–21.
61. Salen P, O’Connor R, Sierzenski P, et al. Can cardiac sonography and capnography be used independently and in combination to predict resuscitation outcomes? Acad Emerg Med 2001; 8:610–15.

5
Stress echocardiography in the emergency room

Eugenio Picano

Key points

- In patients with chest pain, a resting, limited echo (even with a portable unit) offers invaluable information: a newly onset regional wall motion abnormality of ischemic origin can be frequently obvious when electrocardiogram (ECG) changes are absent and cardiac enzymes (including troponin) are not yet abnormal.
- The routine echo can document or raise the suspicion of important nonischemic causes of chest pain, including pericardial effusion, pulmonary embolism, and aortic dissection.
- When resting echo, ECG, and serial enzyme assay are negative, a (physical or pharmacologic) stress echo can be performed even at the bedside. If it is positive, admission to the coronary care unit (in view of ischemia-driven angiography and revascularization) is warranted. If it is negative, the patient can be safely discharged with very low probability of adverse outcome in the short- to medium-term follow-up.

In the USA, approximately 6 million people annually undergo evaluation in the emergency department (ED) for acute chest pain, at a cost of more than $6 billion (Figure 5.1).[1] Most of these patients are admitted to the hospital, and their average length of stay is 1.9 days. Nearly half of the hospitalized patients with unstable angina eventually receive a non-cardiac-related diagnosis[2]—most frequently panic attack,[3] or gastroesophageal reflux or musculoskeletal etiologies.[2] The low-risk patients neither require nor benefit from management in a coronary care unit (CCU). In addition, unnecessary admission to a CCU is inordinately costly, over $2000 per day, and it also reduces the availability of vital CCU beds. Finally, unnecessary CCU admission imposes both undue stress and potential morbidity. Although this low threshold for CCU admission results in a high ratio (4–5:1) of

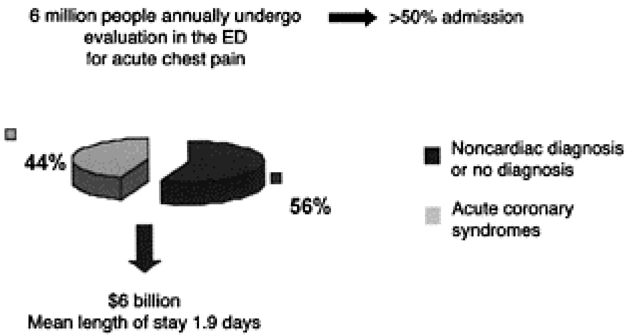

Figure 5.1 The challenge of chest pain. ED: emergency department.

patients with nonischemic causes of chest pain to the number admitted with an ischemic event, 5% of patients with myocardial infarction are inappropriately discharged from the ED. The missed diagnosis accounts for 20% of the claims of indemnity for malpractice in the USA.[2]

In response to this need for improved emergency care, chest pain centers have been established, in which patients are monitored and observed for 24 h. This observation protocol usually incorporates selective historical, physicalexamination, and EGG variables with the concomitant use of serum markers of myocardial cell death (usually creatine kinase-MB or troponin I or T). This primary risk stratification suffers from limitations. Clinical variables have a low specificity, EGG has a high diagnostic accuracy in myocardial infarction but a low accuracy in unstable angina, and biochemical markers of cardiac damage are relatively late.[1,2] In this setting, the incremental value of imaging and stress-test techniques has been documented. Among the imaging techniques, nuclear scintigraphy may accurately image deranged myocardial physiology related to ischemia or infarction, and it has been utilized with some success in patients presenting chest pain.[4] However, scintigraphy is costly, and its implementation in ED patients is limited by both cost and complexity. Nuclear scintigraphy in the emergency department can be a logistical nightmare.[5] The Nuclear Regulatory Commission strictly regulates the use of radioactive isotopes. Emergency physicians may inject the isotopes, provided that they have undergone the necessary radiation training. Furthermore, patients must be removed from the ED to a radioisotope-approved area for the duration of the scan. Rest and stress echocardiography may be easily performed at the bedside in the chest pain unit, and it provides unique information for accurate risk stratification, which is comparable to that of scintigraphic imaging.[5–7]

Resting echocardiography in the ED

Echocardiography has universally recognized advantages over other imaging techniques: lower cost, use of nonionizing radiation, widespread availability, short imaging time, and online interpretation. These advantages are especially important in the ED theater, which

is characterized by logistic restrictions and time constraints. Echocardiography has several critical advantages in chest pain patients, where identification of resting wall motion abnormalities is more sensitive in detecting acute myocardial infarction than the initial EGG. Moreover, the incremental diagnostic value of two-dimensional (2-D) echo is additive to clinical parameters, EGG, and biochemical markers. In addition, a resting 2-D-echo may be essential to diagnose noncoronary (albeit life-threatening) causes of chest pain, including aortic dissection, hypertrophic cardiomyopathy, pulmonary embolism, and pericardial effusion.[8]

However, there are limitations of the 2-D-echo approach in the recognition of myocardial ischemia. The sensitivity is higher in acute myocardial infarction, but lower in unstable angina and non-Q-wave myocardial infarction.[9] This negative finding in the baseline echocardiogram—albeit apparently paradoxical—has clear experimental rationale.[10]

In the experimental animal, the correlation between regional function and flow is continuous, with measurable abnormalities of systolic function for minimal flow reductions. However, the entity of these abnormalities is modest and certainly below the threshold of detection through echocardiography. The clinical manifestation of a regional dysfunction, defined as impairment of wall motion and/or thickening or sufficient severity and/or extension to be detected by 2-D echocardiography, must be associated with a flow reduction greater than 50% in comparison with resting values and involving at least 20% of transmural wall thickness and about 5% of the total myocardial mass. Furthermore, in myocardial infarction, as in transient ischemia, the transmural extent of myocardial damage is correlated with the severity of the regional dyssynergy. A necrosis confined to less than 20% of myocardial thickness is associated with only mild hypokinesia.[10] These experimental data have a clinical correlate: in non-Q myocardial infarction, stable changes of the ST-T segment, with prolonged chest pain and an increase of necrosis enzymes, may be accompanied in 20% of cases by a perfectly normal echocardiogram.[11] Resting echocardiography is therefore very useful in the ED, and it will be more and more used due to technical advances allowing tele-echocardiography with remote consulting,[12] and portable and ultraportable echo instruments allowing user-friendly bedside imaging (see Chapter 6).[13]

Nevertheless, in patients with low-to-intermediate clinical probability of ischemic origin of chest pain, negative baseline echocardiography is not sufficient to rule out acute coronary syndrome, and further action may be required. At that point, a possible strategy is to perform indiscriminate coronary angiography: for patients with low-to-medium probability of disease, this will lead to an enormous escalation of costs, a high number of patients with normal coronary arteries undergoing useless cardiac catheterization.[14] As an alternative, patients may undergo stress testing.[2] Exercise EGG is an excellent option, reasonably safe and with an excellent negative predictive value. However, exercise EGG in these ED patients is often unfeasible and uninterpretable, and the positive predictive value can be suboptimal,[2] due to the recognized limited specificity of the EGG in predicting coronary artery disease.[15] At that point in the flowchart, in patients with still undetermined chest pain, negative serial EGG,

Table 5.1 The prognostic value of stress echo in patients presenting ing with chest pain to the emergency room

Author, year	*Reference No.*	*Stress of choice*	*Patients (n)*	*Mean follow-up (months)*	*Negative predictive value (%)*	*Rate of positivity*
Trippi et al, 1996	16	Dobutamine	139	3	98.5	8/139 (5%)
Colon et al, 1998	17	Exercise	108	12.8	99	8/108 (7%)
Gelejinse et al, 2000	18	Dobutamine	80	6	95	36/80 (45%)
Orlandini et al, 2000	19	Dipyridamole	177	6	99	5/177 (3%)
Buchsbaum et al, 2001	20	Exercise	145	6	99.3	5/145 (3%)
Bholasingh et al, 2003	22	Dobutamine	377	6	96	26/377 (7%)
Bedetti et al, 2004	23	Dipyridamole	552	13±2	98.8	50/552 (9%)
Conti et al, 2005	24	Exercise	503	6	97	99/503 (20%)

negative serial enzymes, and negative baseline echocardiogram, stress echocardiography may represent an excellent option, especially in patients with an exercise EGG that is unfeasible, uninterpretable, or submaximal.

Stress echocardiography in the ED

In the ED, baseline 2-D echocardiography is indispensable, and both echocardiography equipment and expertise must be available at the bedside. This means that when the resting echo is negative the stress echo is already there, at the bedside, with the 2-D-echo machine and the cardiologist interpreting the regional wall motion in real time, and ready to go with the stress echo. Certainly, at that point, the stress chosen may be exercise—but the treadmill introduces additional noise and space requirement into the ED.[8] Pharmacologic stress echo with dobutamine or vasodilators is a more logistically convenient choice, since intravenous access has been previously established upon arrival at the ED. The patient lies in bed in the position most suitable for echo imaging, and the 2-D-echo instrument is already at the bedside (for the resting echocardiogram, whose negativity is a logical prerequisite for further stress testing).

Stress echo has been performed in the ED with several forms of testing,[16–24] including exercise,[17,20,24] dobutamine,[16,18,21,22] and dipyridamole.[19,23] Studies unanimously show a very high feasibility of stress echo—higher with pharmacologic means than with exercise—with an excellent safety profile—better with dipyridamole than with dobutamine—and with very high negative predictive value of stress echo results (Table 5.1). One study reported the similar prognostic accuracy of stress echo and stress SPECT scintigraphy simultaneously performed in the same patient.[24] It is important to note that the rate of positivity in the screened population varied considerably, from 3% to 45%. When the selection criterion is any form of ‘chest pain’, typical or atypical, a very low positivity rate may be expected. If only patients with high-to-intermediate clinical risk are screened, the rate of positivity may be substantially higher.[20] The high and low clinical score may be identified on the basis of chest pain characteristics: location, radiation, character, and associated symptoms (Figure 5.2). The ‘high risk’ corresponds to substernal chest pain, with radiation (arm, shoulder, back, neck, or lower jaw), with character of crushing, pressing, or heaviness, with associated dyspnea and nausea, or with a history of angina. At the opposite end of the clinical spectrum, a Very low’ clinical score corresponds to a pain with apex location, without radiation. The pain may have a sticking, pleuritic, pinprick character, without associated symptoms.[20] The rate of stress echo positivity will be lower if all of the patients with chest pain are evaluated, and higher if only patients with medium-to-high probability on clinical grounds are selected.[21]

Patients with positive stress echo have underlying coronary artery disease and should be admitted to the CCU. The efficiency of this algorithm has been shown not only in single-center experiences but also in large-scale, multicenter validation of the SPEED (Stress Pharmacological Echocardiography in ED) trial, which has analyzed more than 500 patients recruited from six centers of three different countries.[23] The negative predictive value of a negative algorithm (Figure 5.3) is very high (99%). However, there are occasional patients with negative stress test and early readmission for acute coronary syndromes. This may be uncomfortable fo the physician, but is unavoidable if we consider the underlying pathophysiology of the disease. The acute coronary syndromes are linked not only to the anatomical background of hemodynamically critical coronary stenosis—which can be imaged by coronary angiography and represents the physiologic substrate of stress echo positivity—but also to the acute inflammatory phenomena at the vulnerable, not critical plaque, which ‘must’ be missed by stress-testing procedures related to the unmasking of a reduced coronary flow reserve.[25] In particular, a concomitant anti-ischemic therapy at the time of testing should be considered. In the presence of concomitant anti-ischemic therapy, a positive test is prognostically more malignant, and a negative test is prognostically less benign.[26] The currently employed algorithms in the ED certainly minimize the sources of error, but cannot unmask every substrate of myocardial ischemia. The quest for the ‘optimal’ algorithm in the ED will certainly continue in the coming years, but rest and stress echo in the ED are here to stay. They allow an economically convenient, biohazard-free, and ecologically compatible imaging technique,[27] making it possible to lower costs and improve the quality of health care[28] in the critical theater of the ED.

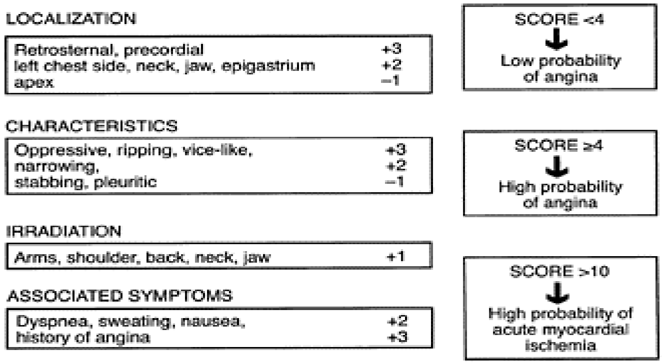

Figure 5.2 Chest pain score in chest pain unit. If only patients with intermediate-to-high score (>4 points) are screened by stress echocardiography, the positivity rate is around 20–30%. If patients with low (<4 points) score are included, the positivity rate may be 10-fold lower. (Reproduced from Conti et al. Am Heart J 2002; 144:630–6.[21] © 2002 with permission from Elsevier.)

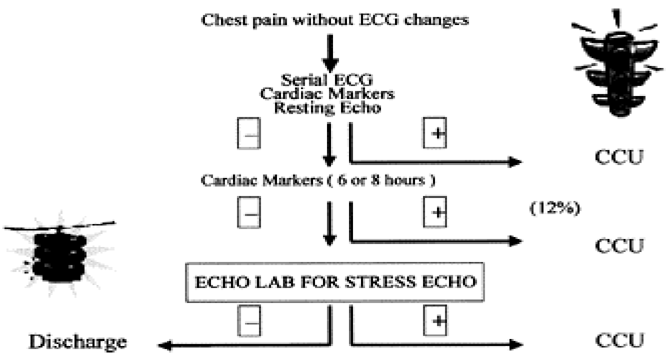

Figure 5.3 The algorithm in the chest pain unit. Patients with negative serial cardiac markers, negative ECG, and negative resting and stress echo can be discharged. Patients positive to at least one of these markers should be

admitted with a high probability of acute coronary syndrome. CCU: coronary care unit.

References

1. Farkouh ME, Smars PA, Reader GS, et al. A clinical trial of a chestpain observation unit for patients with unstable angina. N Engl J Med 1998; 339:1882–8.
2. Stein RA, Chaitman BR, Balady GJ, et al. Safety and utility of exercise testing in emergency room chest pain centers. An advisory from the committee on Exercise, Rehabilitation and Prevention, Council on Clinical Cardiology, American Heart Association. Circulation 2000; 102:1463–7.
3. Carter C, Maddock R, Amsterdam EA, et al. Panic disorder and chest pain in the coronary care unit. Psychosomatics 1992; 33:302–9.
4. Abbott BG, Abdel-Aziz I, Nagula S, Monico EP, Schriver JA, Wackers FJ. Selective use of single-photon emission computed tomography myocardial perfusion imaging in a chest pain center. Am J Cardiol 2001; 87:1351–5.
5. Mather PJ, Shah R. Echocardiography, nuclear scintigraphy, and stress testing in the emergency department evaluation of acute coronary syndrome. Emerg Med Clin North Am 2001; 19:339–49.
6. Ioannidis JP, Salem D, Chew PW, Lau J. Accuracy of imaging technologies in the diagnosis of acute cardiac ischemia in the emergency department: a meta-analysis. Ann Emerg Med 2001; 37:471–7.
7. Kirk JD, Diercks DB, Turnipseed SD, Amsterdam EA. Evaluation of chest pain suspicious for acute coronary syndrome: use of an accelerated diagnostic protocol in a chest pain evaluation unit. Am J Cardiol 2000; 85:40B-48B.
8. Zabalgoitia M, Ismaeil M. Diagnostic and prognostic use of stress echo in acute coronary syndromes including emergency department imaging. Echocardiography 2000; 17:479–93.
9. Picano E. Stress echocardiography: from pathophysiological toy to diagnostic tool. Point of view. Circulation 1992; 85:1604–12.
10. Lieberman AN, Weiss JL, Jugdutt BI, et al. Two-dimensional echocardiography and infarct size: relationship of regional wall motion and thickening to the extent of myocardial infarction in the dog. Circulation 1981; 63:739–50.
11. Carpeggiani C, L’Abbate A, Marzullo P, et al. Multiparametric approach to diagnosis of non-Q-wave acute myocardial infarction. Am J Cardiol 1989; 63:404–8.
12. Trippi JA, Lee KS, Kopp G, Nelson D, Kovacs R. Emergency echocardiography telemedicine: an efficient method to provide 24-hour consultative echocardiography. J Am Coll Cardiol 1996; 27:1748–52.
13. Schiller NB. Hand-held echocardiography: revolution or hassle? J Am Coll Cardiol 2001; 37:2023–4.
14. Kuntz KM, Fleischmann KE, Hunink MG, Douglas PS. Cost-effectiveness of diagnostic strategies for patients with chest pain. Ann Intern Med 1999; 130:709–18.
15. Picano E, Palinkas A, Amyot R. Diagnosis of myocardial ischemia in hypertensive patients. J Hypertens 2001; 19:1177–83.
16. Trippi JA, Lee KS, Kopp G, Nelson DR, Yee KG, Cordell WH. Dobutamine stress tele-echocardiography for evaluation of emergency department patients with chest pain. J Am Coll Cardiol 1997; 30:627–32.

17. Colon PJ 3rd, Guarisco JS, Murgo J, Cheirif J. Utility of stress echocardiography in the triage of patients with atypical chest pain from the emergency department. Am J Cardiol 1998; 82:1282–4.
18. Gelejinse ML, Elhendy A, Kasprzak JD, et al. Safety and prognostic value of early dobutamine-atropine stress echocardiography in patients with spontaneous chest pain and a non-diagnostic electrocardiogram. Eur Heart J 2000; 21:397–405.
19. Orlandini A, Tuero E, Paolasso E, Vilamajo OG, Diaz R. Usefulness of pharmacologic stress echocardiography in a chest pain center. Am J Cardiol 2000; 86:1247–50.
20. Buchsbaum M, Marshall E, Levine B, et al. Emergency department evaluation of chest pain using exercise stress echocardiography. Acad Emerg Med 2001; 8:196–9.
21. Conti A, Paladini B, Toccafondi S, et al. Effectiveness of a multidisciplinary chest pain unit for the assessment of coronary syndromes and risk stratification in the Florence area. Am Heart J 2002; 144:630–5.
22. Bholasingh R, Cornel JH, Kamp O, et al. Prognostic value of predischarge dobutamine stress echocardiography in chest pain patients with a negative cardiac troponin T. J Am Coll Cardiol 2003; 41:596–602.
23. Bedetti G, Pasanisi E, Picano E, et al. Stress echo in chest pain unit: the SPEED (Stress Pharmacological Echocardiography in Emergency Department) trial. Int J Cardiol 2005; 98:754–9.
24. Conti A, Sammicheli L, Gallini C, et al. Assessment of low risk pain patients in the emergency department: head to head comparison of exercise stress echocardiography and exercise myocardial SPECT. Am Heart J 2005; 150:123–7.
25. Sicari R, Cortigiani L, Bigi R, Landi P, Raciti M, Picano E. The prognostic value of pharmacological stress echo is affected by concomitant anti-ischemic therapy at the time of testing. Circulation 2004; 109:2111–16.
26. Picano E, Sicari R. Risk stratification by stress echocardiography: a whiter shade of pale?. Editorial. Eur J Echocardiogr 2004; 5:162–4.
27. Picano E. Stress echocardiography: a historical perspective. Special article. Am J Med 2003; 114;126–30.
28. Picano E. Sustainability of medical imaging. Education and debate. BMJ 2004; 328:578–80.

6
Portable echo in the emergency setting

Satoshi Nakatani and Hiroyuki Kakuchi

Key points

- Portable echo is a powerful tool to assess cardiovascular anatomy, function, and physiology, as in pericardial effusion and tamponade and myocardial infarction, in the emergency setting.
- Wide dissemination of portable echo and use by nonechocardiographers is expected.
- To use this device properly, to obtain accurate data, and to diagnose correctly, proper training is desirable.

Recent advances in electronics and ultrasound technology have enabled the ultrasound industry to create small echocardiography machines, the size of a laptop computer.[1–4] They are basically portable, compact, battery-operated, and inexpensive, and have become a very powerful tool in an emergency setting. Such instruments are primarily designed for focused use, and not for full examinations. However, the important information derived from such portable echocardiography permits faster clinical decisions than waiting for the formal, full echocardiographic examination, and its convenience allows early detection of potentially serious clinical conditions, leading to faster and more accurate treatment.

The terms ‘targeted echo’ and ‘focused echo’ are often used for portable echocardiography. However, we should bear in mind that these words are not the same as ‘incomplete’ or ‘inaccurate’. Even in the emergency setting, we should try to obtain valuable information that leads to accurate diagnosis and determination of the appropriate next steps. We should also avoid inappropriate overuse or underuse of this technology.

Characteristics of portable echocardiography

There are several portable echocardiographic systems on the market. SonoSite (Bothell, WA, USA) offers SonoSite 180 Plus, SonoHeart Elite, SonoSite iLook, and SonoSite Titan. SonoSite iLook is the smallest portable echo (163 mm (H)×266 mm (W)×38 mm (D), weighing 1.4 kg) with a 5-inch monitor, enabling effective hand-held scanning (Figure 6.1A). SonoSite devices have a directional color power Doppler system instead of a conventional color Doppler system to assess blood flow. The directional color power Doppler system can color-code blood flow like a conventional color Doppler system, although no color is assigned for turbulence; blue signifies flow away from the transducer; red, flow toward the transducer (Figure 6.2). The recent models from SonoSite have a capability of pulsed- and continuous-wave Doppler echocardiography, as

previous models did not, enabling assessment of the severity of valvular stenosis and systolic right ventricular pressure. Siemens Medical Systems (Mountain View, CA, USA) offers Cypress (356 mm (H) × 406 mm (W)×343 mm (D), 8.4 kg), which is a ‘full-digital, all-in-one, mobile’ machine equipped with a phased-array transducer (2–4 MHz) (Figure 6.1B). As is shown in the term, ‘all-in-one’, it is capable of second harmonic imaging, contrast imaging, conventional color Doppler, pulsed-wave and continuous-wave Doppler echocardiography, and transesophageal echocardiography, in addition to the usual echocardiographic modes (Figure 6.3). It even has a stress-echocardiography package. OptiGo (Philips Medical Systems, Andover, MA, USA) is a laptop-computer-size (330 mm (H)×89 mm (W)×22.9 mm (D), 3.34 kg) ultrasound system equipped with a 2.5-MHz, phased-array transducer (Figure 6.1C). It

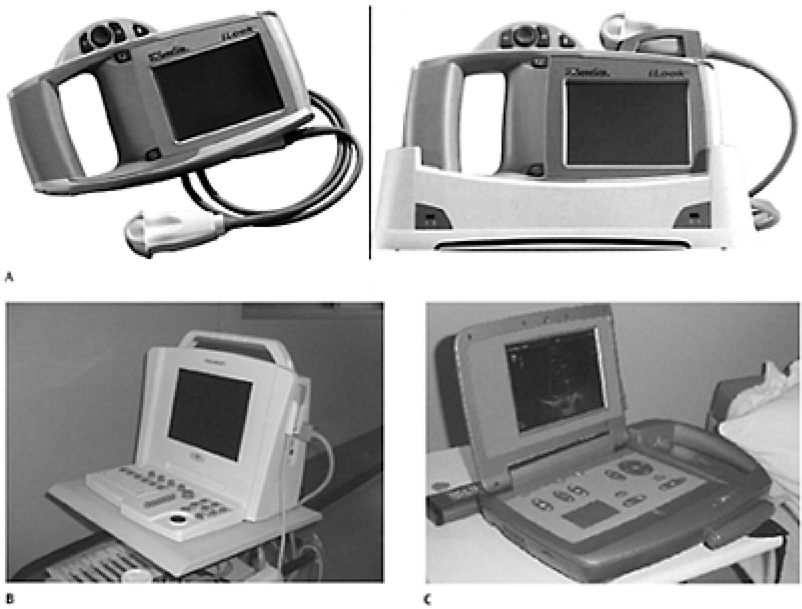

Figure 6.1 Portable echocardiographic machines. SonoSite iLook (A), Cypress (B), and OptiGo (C). The docking station of iLook can be connected to a personal computer to transfer images (courtesy of SonoSite).

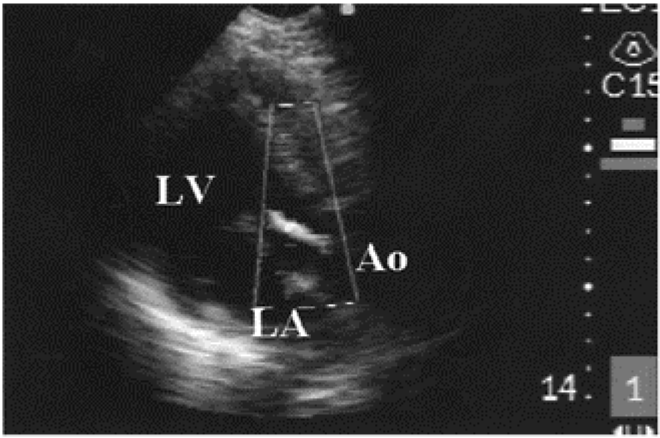

Figure 6.2 Mild aortic regurgitation depicted by directional color power Doppler system. Ao: aorta; LA: left atrium; LV: left ventricle.

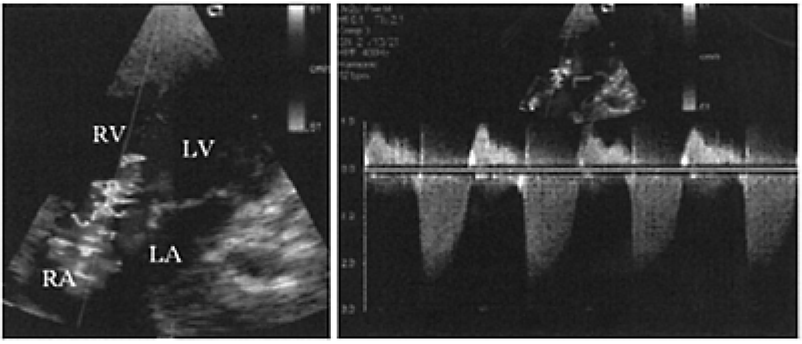

Figure 6.3 Tricuspid regurgitation (left) and its velocity (right) measured by Cypress. LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle.

has a well-designed shape with a transducer holder, enabling good portability.

Role of portable echocardiography in the emergency setting

In the emergency setting, rapid and accurate assessment of cardiovascular anatomy, function, or hemodynamics is always needed. The space for echocardiographic examination, however, is usually limited. Thus, portable echocardiography has a significant role in this setting. There are several reports showing the value of portable echocardiography in rapid assessment of cardiac function and anatomy in acute settings.[5,6] We determined the clinical usefulness of portable echocardiography in 57 patients who visited our cardiac emergency department. We performed quick portable echocardiographic examination after taking history, monitoring vital signs, and administering electrocardiographic examination followed by echocardiography with a high-end machine. Overall, we could reach a correct diagnosis in 95% of all cases by portable echocardiography. Figure 6.4 shows a left ventricular apical thrombus associated with anteroapical myocardial infarction. This patient came to our emergency room with a complaint of chest pain that he had noted 3 days before. When we scanned this patient in a routine examination in the emergency room, we found a big mobile thrombus in the left ventricular apex. Therefore, we admitted him to our coronary care unit immediately, and we could quickly start appropriate treatment. The apical thrombus disappeared in a week without any events of systemic embolization. We were able to detect causes of chest or abdominal pain of even extracardiac origin, such as dilated renal pelvis due to a urinary tract calculus, pleural effusions, gallstone, and ileus. Thus, portable echocardiography seems to be useful to triage or screen patients in an emergency setting.

Portable echocardiography is also very useful to guide a needle placement for pleural or pericardial fluid (see Chapter 2). We do not have to carry a big ultrasound machine to a small patient’s room. We can confirm the presence and amount of fluid, determine the right position and direction of needle insertion, confirm the correct position of the tip of a needle by injecting a small amount of agitated saline, monitor the drainage continuously, and, finally, evaluate the cardiac function just after drainage (Figure 6.5).

The other situation in which we use portable echocardiography is a cardiac catheterization room where a big ultrasound machine is not fit. In percutaneous transvenous balloon mitral valvuloplasty, it can guide a transseptal needle insertion and assess the mitral valve morphology and regurgitation before and just after valvuloplasty (Figure 6.6). Portable echocardiography is also used during percutaneous transluminal septal myocardial ablation for hypertrophic obstructive cardiomyopathy. Estimation of the size of the septal vascular territory with myocardial contrast echocardiography has been useful in

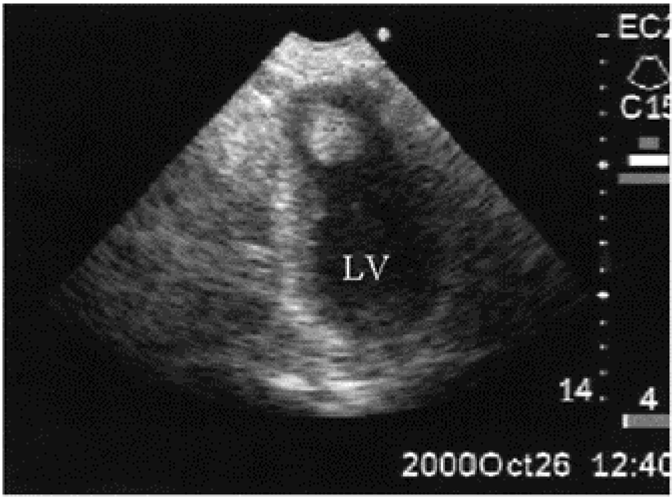

Figure 6.4 Left ventricular apical thrombus depicted by SonoHeart. This patient was admitted to the coronary care unit after SonoHeart scanning. LV: left ventricle.

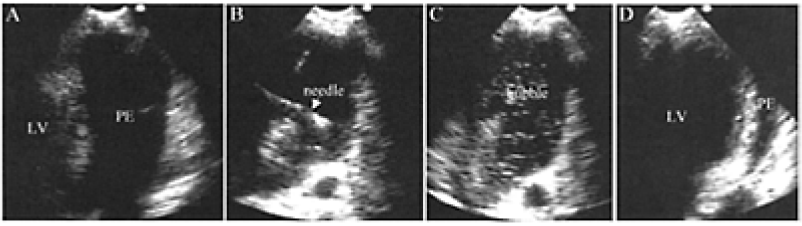

Figure 6.5 Portable echocardiography during pericardiocentesis. (A) A large amont of pericardial effusion (PE) was seen. (B) Under guidance of SonoHeart, a needle was inserted into the pericardial space. (C) To determine the location of the needle, a small amount of agitated saline was injected through the needle and the bubbles were seen. (D) After drainage, the amount of pericardial effusion was

significantly reduced. LV: left ventricle.

successful septal myocardial ablation.[7] We use the device to do contrast echocardiography before alcohol injection to confirm proper localization of the catheter tip.

Who should use portable echo?

Complete and comprehensive echocardiographic examination is not always necessary in the emergency setting. Rather, echocardiography is often used to probe specific problems as in the assessment of cardiac function, the presence or absence of pericardial effusion, aortic dissection, and so on. Since this device is portable, it can be used as a tool for ‘ultrasound-assisted physical examination’. This means any personnel in the emergency room can use this device. Cardiology trainees or even medical students can use portable echocardiography for a point-of-care must be aware that the information derived from portable cardiac examination after proper training.[8,9] However, we echocardiography should be interpreted correctly or should not be interpreted by untrained individuals, because poor imaging techniques may result in inaccurate diagnosis. The user of portable echocardiography should

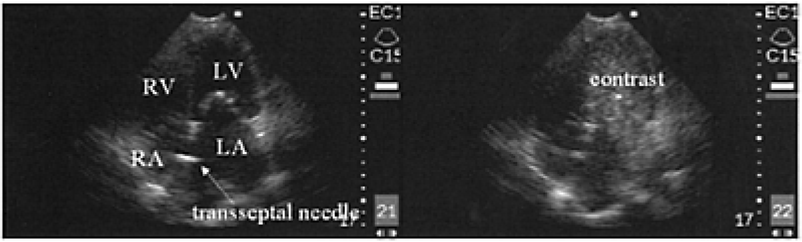

Figure 6.6 Portable echocardiography during percutaneous transvenous balloon mitral valvuloplasty. Left panel: a transseptal needle is seen across the atrial septum. Right panel: a small amont of agitated contrast media is injected from a transseptal catheter to confirm the position of its tip. Contrast echo is seen in the left atrium and flows to the left ventricle, showing the correct positioning of the catheter.

LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle.

take personal responsibility for the image and information obtained from the device and should determine which image or information is important to answer the specific clinical questions. Therefore, the user should be experienced with ordinary echocardiography or should have appropriate training for the use of the device and for interpretation data obtained from portable echocardiography.

The American Society of Echocardiography has published a special report on recommendations regarding portable echocardiography.[10] In this report, portable echocardiography has been considered to extend the concept of the Complete physical examination', allowing more rapid assessment of cardiovascular anatomy and hemodynamics than with a high-end ultrasound machine. With wide dissemination of portable echocardiography, it is speculated that a diverse group of users, including nonechocardiographers, will be involved. Therefore, to avoid inappropriate use, misapplication, or even misdiag nosis, the society recommends proper user-specific training. The society considers that level 1 of training may not be adequate for the independent performance and interpretation of a comprehensive clinical echocardiographic examination (Table 6.1).[10] For the application of portable echocardiography in the emergency setting, level 2 or 3 of training is mandatory.

Limitations of portable echo

In one paper,[6] the limitations of portable echocardiography in assessing critically ill patients was demonstrated. The authors compared the results obtained from portable echocardiography with those obtained from a high-end machine in 80 critically ill patients. They found one or more missed findings by portable echocardiography in

Table 6.1 Minimum recommended levels of training in echocardiography[10]

Level	*Total number of personally performed examinations*	*Total number of personally interpreted examinations*	*Objectives*	*On completion*
1	75	150	Introductory experience	Perform with supervision
2	150	300	Performance, interpretation	Perform independently
3	300	700	Laboratory director and teacher	

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45% of the patients. The causes of this unexpectedly high percentage included the lack of spectral Doppler, M-mode echocardiography, electrocardiogram, and color flow Doppler imaging in the study device. Using the same system, we also missed some clinical findings related to the reason for referral to the emergency setting. Overall, further examination was required in 33% of our patients. They included patients with congestive heart failure requiring continuous-wave Doppler echocardiography to estimate pulmonary hypertension and a patient with suspected pulmonary thromboembolism requiring assessment of pulmonary hypertension and lung perfusion scintigraphy. However, those limitations are now mostly overcome, and much better results are expected with the latest system.

The recent development of portable echocardiography has been remarkable and very useful in screening or triaging patients in an emergency setting. In most cases, portable echocardiography provides us with necessary information to reach a correct diagnosis. However, it sometimes gives us only suboptimal images and incomplete information. Therefore, if patients show abnormal findings that should be pursued more, they should be referred for complete echocardiographic evaluation, which would provide better image quality and complete hemodynamic evaluation.

Future perspective

It seems that the development of ultrasound equipment is moving in two different directions. One is toward the development of a fully equipped high-end machine. It would perform recent and cutting-edge techniques, such as strain Doppler echocardiography and contrast echocardiography, as well as routine echocardiography. The other is toward the development of a hand-held ultrasound scanner. Because of its size, convenience, and the information that it provides, the hand-held scanner is very useful in the emergency setting and will become more common in the future. A recent paper has reported a unique application of portable echocardiography.[11] The authors successfully transmitted echocardiograms obtained by portable echocardiography in an ambulance to a tertiary-care center, providing accurate evaluation of left ventricular size and function and the presence of pericardial effusion before arriving at the center.

The hand-held scanner is becoming smaller and smaller, and it will soon be of personal digital assistant (PDA) size. Physicians will use it like a stethoscope anywhere and anytime they need it. This means that this type of hand-held scanner will be used not only by cardiologists but also by internists, emergency physicians, vascular physicians, surgeons, and radiologists. However, one should bear in mind that a physician who uses a hand-held scanner is not always an echocardiologist. Therefore, we believe that anyone that wants to use it should have appropriate training in cardiology, echocardiography, and abdominal or vessel ultrasound for its efficient use in improvement of patient care and outcome.

References

1. Bruce CJ, Montgomery SC, Bailey KR, Tajik J, Seward B. Utility of hand-carried ultrasound devices used by cardiologists with and without significant echocardiographic experience in the cardiology inpatient and outpatient settings. Am J Cardiol 2002; 90:1273–5.
2. Popp RL. Perspective—the physical examination of the future: echocardiography as part of the assessment. ACC Curr J Rev 1998; 7:79–81.
3. Kimura BJ, Prezeshki B, Frack SA, DeMaria AN. Feasibility of ‘limited’ echo imaging: characterization of incidental findings. J Am Soc Echocardiogr 1998; 11:746–50.
4. Spencer KT, Anderson AS, Bhargava A, et al. Physician-performed point-of-care echocardiography using a laptop platform compared with physical examination in the cardiovascular patient. J Am Coll Cardiol 2001; 37:2013–18.
5. Rugolotto M, Chang CP, Hu B, Schnittger I, Liang DH. Clinical use of cardiac ultrasound performed with a hand-carried device in patients admitted for acute cardiac care. Am J Cardiol 2002; 90:1040–2.
6. Goodkin GM, Spevack DM, Tunick PA, Kronzon I. How useful is hand-carried echocardiography in critically ill patients? J Am Coll Cardiol 2001; 37:2019–22.
7. Nagueh SF, Lakkis NM, He ZX, et al. Role of myocardial contrast echocardiography during nonsurgical septal reduction therapy for hypertrophic obstructive cardiomyopathy. J Am Coll Cardiol 1998; 32:225–9.
8. Lemola K, Yamada E, Jagasia D, Kerber RE. A hand-carried personal ultrasound device for rapid evaluation of left ventricular function: use after limited echo training. Echocardiography 2003; 20:309–12.
9. Duvall WL, Croft LB, Goldman ME. Can hand-carried ultrasound devices be extended for use by the noncardiology medical community? Echocardiography 2003; 20:471–6.
10. Seward JB, Douglas PS, Erbel R, et al. Hand-carried cardiac ultrasound (HCU) device: recommendations regarding new technology. A report from the echocardiography task force on new technology of the nomenclature and standards committee of the American Society of Echocardiography. J Am Soc Echocardiogr 2002; 15:369–73.
11. Garret PD, Boyd SYN, Bauch TD, et al. Feasibility of real-time echocardiographic evaluation during patient transport. J Am Soc Echocardiogr 2003; 16:197–201.

7
Echocardiography in detecting cardiac sources of embolism

Satoshi Nakatani

Key points

- Transesophageal echocardiography (TEE) should be considered in patients with stroke.
- Left atrial appendage should be viewed from multiple planes.
- Vegetations should be searched by TEE.
- Contrast echocardiography under the Valsalva maneuver should be performed in a patient suspected of paradoxical embolism.

Stroke is one of the leading causes of death and disability in the developed countries. Detection of sources of embolism is important to understand the cause of stroke and to prevent further stroke attacks. Since the heart is often the origin of an occlusive embolus with subsequent migration to a target organ, imaging of the heart is clinically valuable. Several modalities, including cardiac computed tomography (CT) and magnetic resonance imaging (MRI), are used to identify cardiac embolic sources. However, transthoracic echocardiography and transesophageal echocardiography (TEE) are the most commonly used among them because of their high diagnostic capabilities and low invasiveness.[1] This has been clarified in the ACC/AHA guidelines that consider echocardiography as an appropriate tool for examining patients with embolic events.[2] Table 7.1 shows the indications for echocardiography in patients with neurologic events or other vascular occlusive events shown in the guidelines.

Thrombus in the left atrium

The left atrium is the most common site of thrombus in patients with stroke. Especially, dilation combined with atrial fibrillation promotes thrombus formation in the left atrium. Left atrial dilation is seen in many disorders such as rheumatic mitral valve disease, valvular and nonvalvular atrial fibrillation, and cardiomyopathy. Generally speaking, transthoracic echocardiography well visualizes left atrial cavity, especially with harmonic imaging (Figure 7.1). However, transthoracic echocardiography is not often

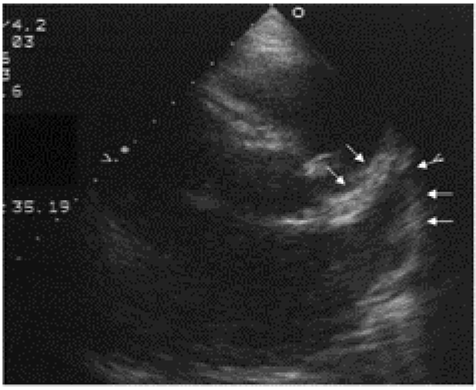


Figure 7.1 Left atrial appendage (arrows) depicted by transthoracic echocardiography.

Table 7.1 Indications for echocardiography in patients with neurologic events or other vascular occlusive events

Occlusive events	*Class*
1. Patients of any age with abrupt occlusion of a major peripheral or visceral artery	I
2. Younger patients (typically <45 years) with cerebrovascular events	I
3. Older patients (typically>45 years) with neurologic events without evidence of cerebrovascular disease or other obvious cause	I
4. Patients for whom a clinical therapeutic decision (such as anticoagulation) depends on the results of echocardiography	I
5. Patients with suspicion of embolic disease and with cerebrovascular disease of questionable significance	IIa
6. Patients with a neurologic event and intrinsic cerebrovascular disease of a nature sufficient to cause the clinical event	IIb
7. Patients for whom the results of echocardiography will not affect the decision to institute anticoagulant therapy or otherwise alter the approach to diagnosis or treatment	III

Class I. Conditions for which there is evidence and/or general agreement that a given procedure of treatment is useful and effective
Class II. Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment
IIa: Weight of evidence/opinion is in favor of usefulness/efficacy
IIb: Usefulness/efficacy is less well established by evidence/opinion
Class III. Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/effective and in some cases may be harmful

satisfactory in detecting left atrial thrombus, especially when the thrombus is in the left atrial appendage. TEE is much superior to the transthoracic echocardiography in detection of thrombus in the left atrium. Figure 7.2 shows a thrombus in the left atrial appendage in a patient with stroke. This patient has a dilated left atrium due to rheumatic mitral stenosis and atrial fibrillation, which are apparently risk factors for left atrial thrombus.

The shape of the left atrial appendage varies and sometimes the appendage has multilobes.[3] Veinot et al have shown from 500 normal autopsy hearts that the most frequent occurrence was a two-lobe left atrial appendage (54%), but a three- or four-lobe appendage was also common (26%).[3] In order not to miss even a tiny thrombus in the left atrial appendage, which has such a complicated shape, we should depict the whole image of the appendage

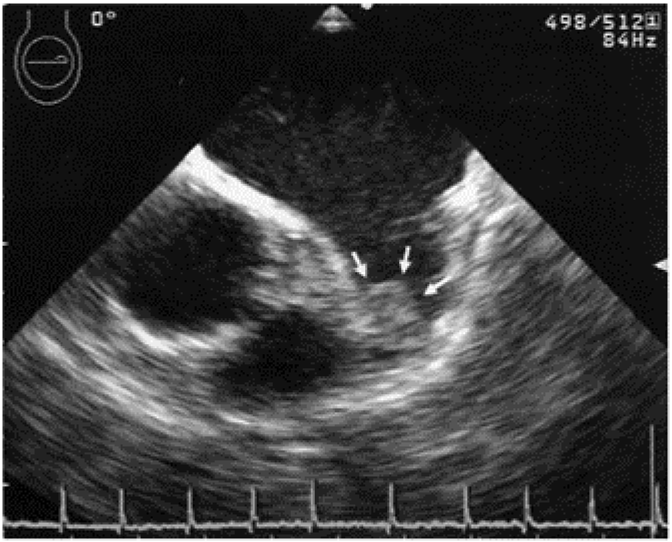


Figure 7.2 Thrombus in the left atrial appendage (arrows) demonstrated by transesophageal echocardiography.

from multiple views by multiplane TEE if available (Figure 7.3). This procedure also helps differentiate actual thrombus from pectinate muscles (Figure 7.4) or echo artifact.

Left atrial appendage flow velocities, easily obtained bypulsed-wave Doppler technique, reflect appendage function and are significant predictors of thrombus

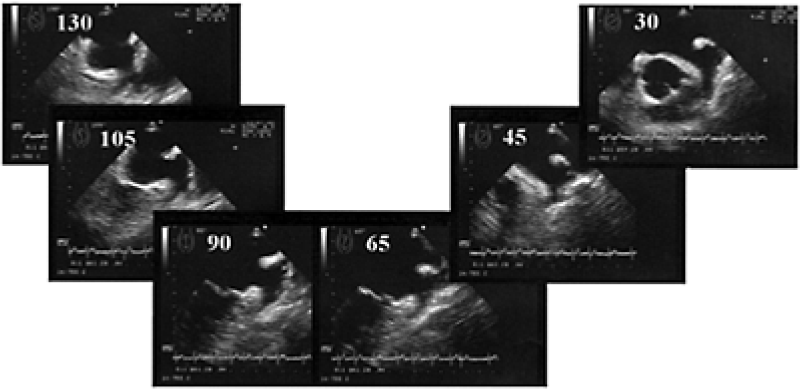


Figure 7.3 Left atrial appendage shown from multiple views by multiplane transesophageal echocardiography. Numbers in the upper left corner indicate rotation degrees of multiplane probe.

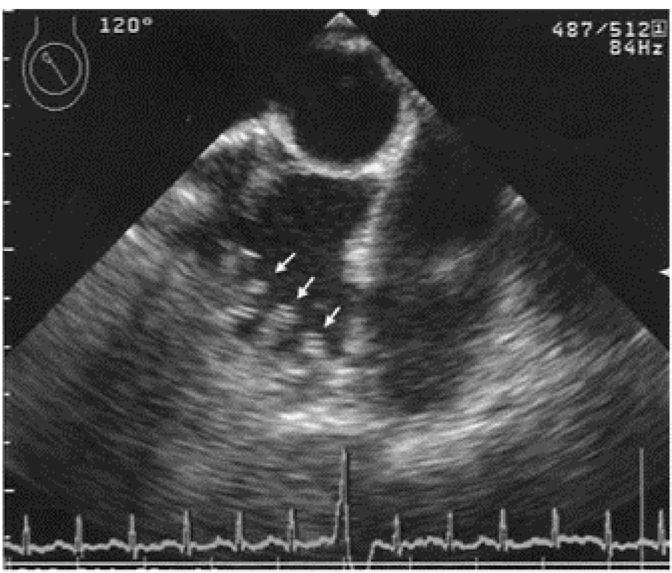


Figure 7.4 Pectinate muscles (arrows) demonstrated by transesophageal echocardiography viewed at 120°.

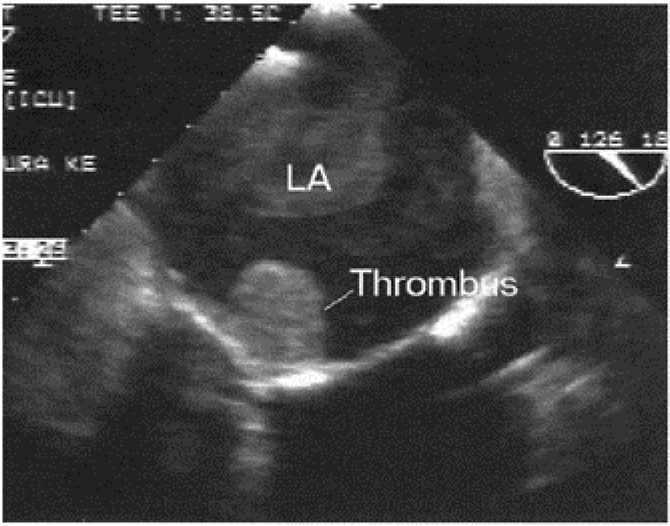


Figure 7.5 Thrombus seated on the mitral prosthetic valve ring demonstrated by transesophageal echocardiography. Significant spontaneous echocardiographic contrast is seen in the left atrium (LA).

formation. It has been reported that the mean emptying velocity of the left atrial appendage is 50–60 cm/s, and the mean filling velocity is 40–50 cm/s in patients without cardiac abnormalities.[4] The low flow velocities have been associated with predisposition to thrombus formation in the left atrial appendage. In the SPAF III trial, appendage thrombi were more prevalent in patients with low flow velocities of ≤20 cm/s than in those with higher velocities.[5] Left atrial appendage thrombus is sometimes difficult to detect in normal structure such as pectinate muscles even with multiplane TEE. On such occasions, the presence of low flow velocities supports a diagnosis of appendage thrombus, whereas normal function suggests an alternative diagnosis. Many studies have shown an association between appendage dysfunction and previous stroke. The relative risk of stroke has been reported 2.6 times in patients with low flow velocities compared to those with normal flow velocities.[5] The other sign of left atrial appendage dysfunction is the presence of spontaneous echocardiographic contrast. It has been shown that spontaneous echocardiographic contrast was seen in 75% of patients with left atrial appendage-emptying velocity of less than 20 cm/s. Moreover, prevalence of neurologic events was much higher in patients with spontaneous echocardiographic contrast (20.5%) than in those without contrast (5.7%).[2,6] Spontaneous echocardiographic contrast is basically rouleax formation of red blood cells and implies blood stasis. However, we should note that its presence may be sensitive but is not a specific sign of thrombus formation.

Thrombus can be found in the left atrial body as well as in the left atrial appendage, although the latter is the most common site of the thrombus formation. Left atrial body thrombus is not found in places where pulmonary venous flow or mitral regurgitation flow washes out. Rarely, floating ball thrombus is found in the left atrial cavity, usually requiring urgent surgery.

In cases with thrombosed prosthetic valves, especially those in the mitral position, thrombi are often hard to find by transthoracic echocardiography. Of course, transthoracic echocardiography can detect abnormal transmitral pressure gradient suggesting prosthetic valve obstruction or mitral regurgitation, but the cause of the prosthetic valve dysfunction is often undefined. In such disorders, TEE is essential since it can well visualize the mitral valve from an atrial aspect without interference by acoustic shadow from the prosthetic material (Figure 7.5).

Thrombus in the left ventricle

In the event of stagnant blood flow in the left ventricle, which might occur after a myocardial infarction or in the setting of dilated cardiomyopathy, thrombus can be found in the left ventricle. Anterior myocardial infarction has a higher rate of left ventricular thrombus than inferior myocardial infarction.[7] Figure 7.6 shows left ventricular thrombus in the apex in a patient with anterior myocardial infarction. As shown in this figure, transthoracic

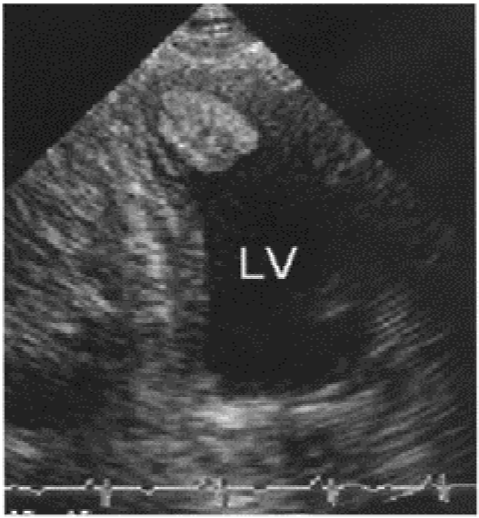


Figure 7.6 Left ventricular apical thrombus found in a patient with anterior myocardial infarction. LV: left ventricle.

echocardiography is capable of determining left ventricular thrombus. Unlike in left atrial thrombus, TEE is not a powerful tool to depict ventricular thrombus. This is mainly because of the distance from the transducer to the thrombus and the limited views of the transesophageal approach. It is not always so easy to look at the left ventricular apex by TEE because routine midesophageal views foreshorten the left ventricle. Deep transgastric views may be useful in visualizing the apex. Dilated cardiomyopathy is another situation of left ventricular apical thrombus. Since anticoagulation is effective in reducing the rate of embolization produced by left ventricular thrombus, this should be considered in patients with broad anterior or anteroapical myocardial infarction or profound left ventricular systolic dysfunction.

Another condition accompanied by left ventricular thrombus is hypereosinophilic syndrome (Löffler's endocarditis). Cardiac involvement, such as endomyocardial fibrosis, restrictive filling, and left ventricular apical thrombus, is seen in approximately 40–75% of idiopathic hypereosinophilic syndrome.[8] Since left ventricular thrombus in hypereosinophilic syndrome is based on the injured endocardium, it can be formed in the area of preserved wall motion. Thus, in a patient with hypereosinophilic syndrome, we should discard the notion that thrombus cannot be formed in the area of preserved wall motion. Figure 7.7 shows a typical apical mural thrombus in hypereosinophilic syndrome.[9]

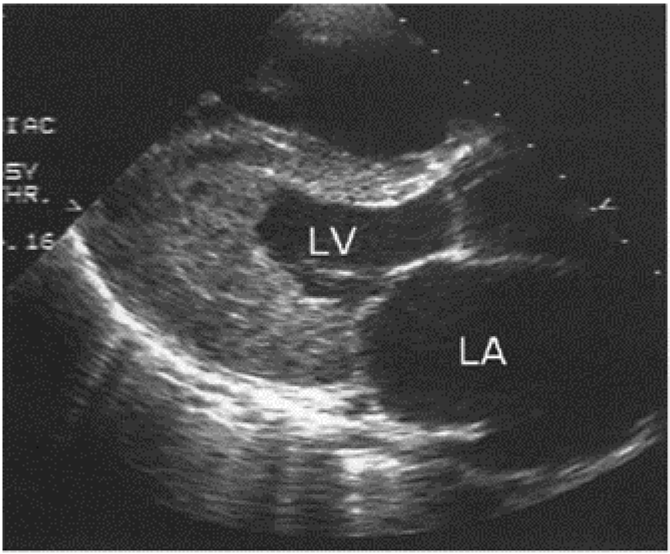


Figure 7.7 Left ventricular apical mural thrombus found in a patient with hypereosinophilic syndrome. LA: left atrium; LV: left ventricle.

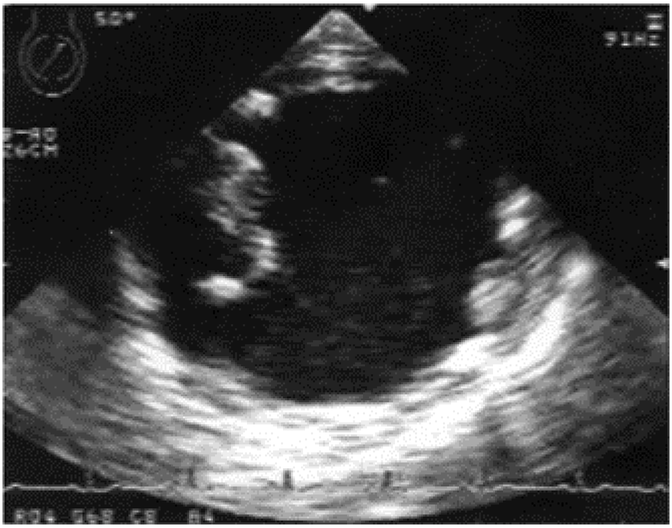


Figure 7.8 Mobile and protruding aortic atheromas depicted by transesophageal echocardiography.

Thrombus or atheroma in the aorta

It is assumed that either thrombus on the plaque or plaque material itself in the aorta breaks off and causes systemic embolization. These atheroemboli typically occlude small downstream arteries and arterioles. When these are in the aortic arch, they may cause stroke.

TEE is quite useful to visualize with clarity the ascending aorta and the proximal aortic arch beside the descending aorta; this cannot be achieved with transthoracic echocardiography. It has been shown that protruding aortic atheromas are detected in 7% of patients during routine TEE, and about one-third of those patients have embolic episodes during a 2-year follow-up period.[10] The embolic risk is even higher in patients with pedunculated or mobile lesions or invasive procedures.[11] Mobile atheroma was found in 0.3% of control subjects and in 6.8% of patients with cerebrovascular events (Figure 7.8).[12,13] The size of atheroma plaque seems also to be an important predictor of embolic events. Amarenco et al found atherosclerotic plaque of 4 mm or greater in the ascending aorta or proximal arch in 14% of patients with stroke, but in only 2% of controls.[13] These previous studies have taught us that, in a patient with an embolic event, the thoracic aorta should be investigated as a potential source by TEE, and when a large, protruding, or pedunculated atheromatous plaque or thrombus is identified, an invasive aortic procedure should be avoided if possible.

Vegetations

Vegetations found in patients with infective endocarditis can embolize, causing stroke in more than 15% of patients. Echocardiography is an essential tool to identify vegetations (Figure 7.9). The sensitivity of detecting vegetations is approximately 60% by transthoracic echocardiography and that by TEE is approximately 90%. The value of TEE is much enhanced in patients with prosthetic valve endocarditis. Abnormal prosthetic valves with vegetations, especially those in the mitral position, are much better evaluated by the transesophageal approach than the transthoracic approach. Transthoracic echocardiography is not suitable to visualize vegetations seating on the atrial aspect of mitral prosthesis because of the echo-reflective properties of prosthetic material. Therefore, virtually all patients clinically suspected of infective endocarditis should undergo TEE. It has been shown that the size (>10–15 mm) and mobility of vegetations are important factors to predict ischemic stroke events.[14,15] Sanfilippo et al classified vegetation by its size, mobility, extent, and consistency or texture.[14] They found that vegetation size, site, extent, and mobility were important predictors of complications, including stroke, and an echocardiographic score based on these parameters could predict the occurrence of complications with 70% sensitivity and 92% specificity in mitral valve endocarditis, and with 76% sensitivity and 62% specificity in aortic valve endocarditis. A recent investigation has focused on only

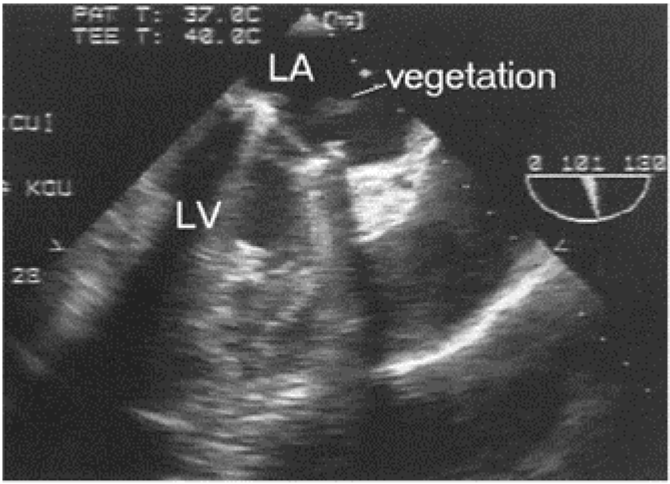


Figure 7.9 Vegetation attached to the mitral prosthetic valve ring demonstrated by transesophageal echocardiography. LA: left atrium; LV: left ventricle.

the embolic events and attempted to determine which TEE factors were most related to embolism in patients with infective endocarditis.[15] They have shown that a significantly higher incidence of embolism was present in patients with vegetation length more than 10 mm (60%) and in patients with mobile vegetations (62%). In patients who had both severely mobile and large vegetations (>15 mm), embolism was particularly frequent, occurring in 83% of such patients (Figure 7.10). The rate of embolic events falls when appropriate antibiotics are effective. However, once embolism occurs, the disorder frequently becomes fatal. Therefore, in patients with vegetations over 15 mm and high mobility, early operation may be recommended, irrespective of the degree of valve destruction, heart failure, and response to antibiotic therapy, although there have been no studies demonstrating an early operation changes outcome.

Cardiac tumors

Cardiac tumors can cause stroke. The most common tumor in the heart is myxoma, comprising 50% of all primary cardiac tumors.[16] Myxoma often arises from the atrial septum. Transthoracic echocardiography is often adequate to investigate atrial myxoma. However, TEE provides better images than the transthoracic approach and much more information, such as the accurate shape and consistency, and the attached site. An embolic event is one of the first presentations of cardiac myxoma, occur-

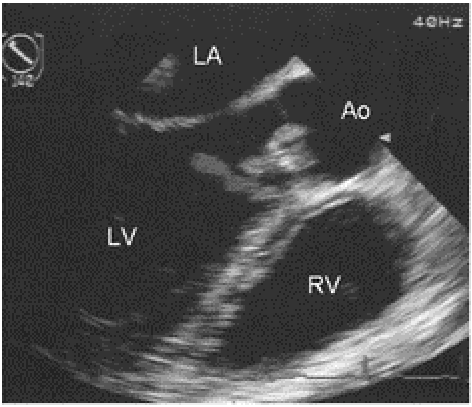


Figure 7.10 Native valve endocarditis with large and mobile vegetations attached to the left ventricular outflow tract and the aortic valve. Ao: aorta; LA: left atrium; LV: left ventricle; RV: right ventricle.

ring in 30% of patients with this diagnosis. In general, the mobility and friability of a tumor and tendency for thrombus formation may relate to propensity to embolization.

One paper has reported that the incidence of embolism is significantly higher in the polypoid type (soft and irregular shape with mobile surface) than in the

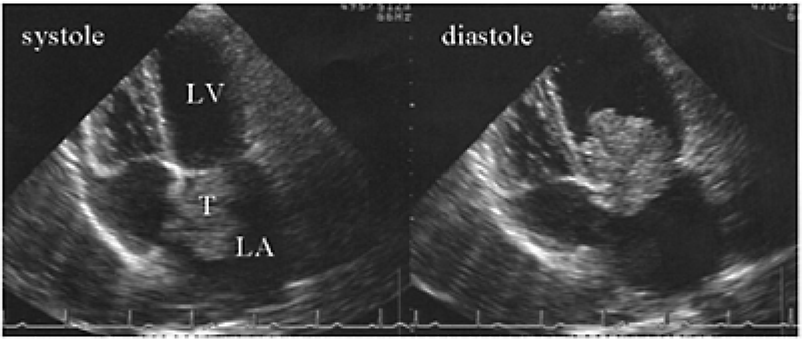


Figure 7.11 Polypoid type myxoma attached to the atrial septum. LA: left atrium; LV: left ventricle; T: myxoma.

round type (solid and round shape with nonmobile surface) (58% versus 0%, $P<0.05$).[17] Figure 7.11 shows one example of the polypoid type of myxoma. Other benign tumors, such as papillary fibroelastoma, and malignant tumors, such as sarcoma, can cause embolization as well.

Papillary fibroelastoma is mostly found on the valvular endocardium but can be found anywhere in the heart. Histologically, papillary fibroelastoma has a central core of dense connective tissue surrounded by an acid mucopolysaccharide matrix, smooth muscle cells, and collagen, and it is covered by endothelium. On gross inspection, such tumors are usually small and have a characteristic frondlike appearance, resembling a sea anemone (Figure 7.12). Although papillary fibroelastoma occurs in all age groups, it is most commonly seen in the aortic or mitral valve of elderly patients. This supports the hypothesis that papillary fibroelastoma may represent a degenerative process. Papillary fibroelastoma is clinically silent, but it, or the thrombus attached to it, has the potential to cause embolism. Papillary fibroelastomas have been operatively excised in patients with evidence of stroke.[18] However, when they are detected in asymptomatic patients, operation solely for its excision is rarely indicated.

Paradoxical embolism

Emboli arising in systemic veins may pass directly to the systemic circulation when there is right-to-left intracardiac

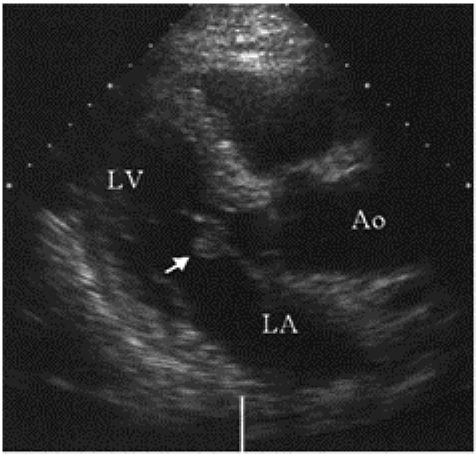


Figure 7.12 A tumor found on mitral leaflet in an asymptomatic patient (arrow). Since the echocardiographic appearance looks like a sea anemone, this was considered papillary fibroelastoma. Ao: aorta; LA: left atrium; LV: left ventricle.

shunting. This is called paradoxical embolism. Color Doppler echocardiography can demonstrate shunting flow, but intravenous injection of a contrast agent, such as agitated saline, can detect more sensitively right-to-left

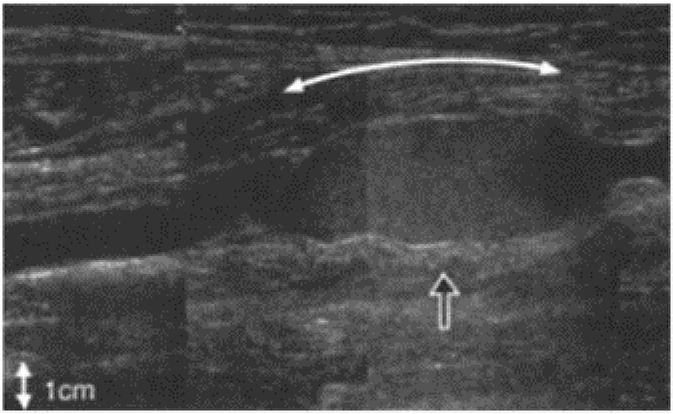


Figure 7.13 Big thrombi (arrow) seated in the soleus vein.

shunting across a patent foramen ovale. Recent studies have reported the link between a patent foramen ovale and embolic events. In the control subjects, a patent foramen ovale was found in 10%, but in patients with cryptogenic embolism it was found in more than 30% by echocardiography.2,19–21 At autopsy, it has been found in 25–30% of normal subjects.[22] The diameter of a patent foramen ovale has been shown to be associated with the risk of embolic events.[23] Paradoxical embolism is sometimes seen in patients with cyanotic heart disease. However, even if there is no cyanotic heart disease, paradoxical embolism could occur under the condition of transient elevation of right heart pressure. A small amount of right-to-left shunting in patients without atrial septal defect can be demonstrated by the Valsalva maneuver and an intravenous contrast agent such as agitated saline. One study has shown that, with the Valsalva maneuver and contrast echocardiography, 18% of normal subjects exhibit transient right-to-left shunting. Moreover, 5% of normal subjects showed shunting even at rest.[24] This indicates that paradoxical embolism could occur under physiologic conditions. Therefore, in a patient with stroke whose embolic source is uncertain, the Valsalva maneuver with contrast agent should be monitored by transthoracic echocardiography or TEE. To differentiate shunting from transpulmonary passage of bubbles, we usually consider as diagnostic only bubbles that can be observed to cross the atrial septum or appear in the left atrium within three heart cycles from contrast appearance in the right heart.

Ultrasound scanning of the peripheral veins gives us important information on emboli in systemic veins. Figure 7.13 shows a big thrombus found in the soleus vein. Not only cardiac ultrasound but also peripheral ultrasound could be a technique for cardiologists to learn.

Atrial septal aneurysm

Atrial septal aneurysm, the redundancy of atrial septum, is a well-recognized cardiac abnormality found in around 2% of the population, and it may be associated with cardiac embolism. There are several diagnostic criteria for atrial septal aneurysm. By one criterion, it is diagnosed if the atrial septum or part of it exhibits aneurysmal dilation protruding at least 1.5 cm beyond the plane of the atrial septum, or if it exhibits phasic excursion during the cardiorespiratory cycle exceeding 1.5 cm and if the base of the aneurysmal protrusion is at least 1.5 cm in diameter.[25] Another criterion stipulates 1.0 instead of 1.5 cm. TEE is superior to the transthoracic approach in the diagnosis of atrial septal aneurysm. Atrial septal aneurysm can be formed secondary to interatrial pressure differences but may also be a primary malformation involving the region of the fossa ovalis or the entire septum.

Several reports have suggested its association with cardiogenic embolism. The incidence of embolism in patients with atrial septal aneurysm is 20–50%.[26] However, the mechanism of its association with embolism is uncertain. Thrombus may not be formed on the constantly moving atrial septum but may be formed in the ‘tunnel-like’ region of the aneurysm and atrial septum. Atrial septal aneurysm is often associated with other cardiac abnormalities, such as atrial septal defects or mitral valve prolapse. In a large series of cases with atrial septal aneurysm, about half of patients showed interatrial shunting and about 40% of patients had episodes of possible cardiogenic embolism.[27]

Thus, it has been thought that interatrial shunting may be a reason for the association of atrial septal aneurysm and embolism.

In summary, there are numerous cardiac etiologies for the source of an embolus. When therapeutic options vary by etiology, echocardiography, especially TEE, can provide the necessary information for proper management.

References

1. McNamara RL, Lima JA, Whelton PK, Powe NR. Echocardiographic identification of cardiovascular sources of emboli to guide clinical management of stroke: a cost-effectiveness analysis. Ann Intern Med 1997; 127:775–87.
2. Cheitlin MD, Alpert JS, Armstrong WF, et al. ACC/AHA guidelines for the clinical application of echocardiography: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Application of Echocardiography). Circulation 1997; 95:1686–744.
3. Veinot JP, Harrity PJ, Gentile F, et al. Anatomy of the normal left atrial appendage. A quantitative study of age-related changes in 500 autopsy hearts: implications for echocardiographic examination. Circulation 1997; 96:3112–15.
4. Agmon Y, Khandheria BK, Gentile F, Seward JB. Echocardiographic assessment of the left atrial appendage. J Am Coll Cardiol 1999; 34:1867–77.
5. Stroke Prevention in Atrial Fibrillation Investigators Committee on Echocardiography. Transesophageal echocardiographic correlates of thromboembolism in high-risk patients with nonvalvular atrial fibrillation. Ann Intern Med 1998; 128:639–47.
6. Leung DY, Black IW, Cranney GB, Hopkins AP, Walsh WF. Prognostic implications of left atrial spontaneous echo contrast in nonvalvular atrial fibrillation. J Am Coll Cardiol 1994; 24:755–62.
7. Vaitkus PT, Barnathan ES. Embolic potential, prevention and management of mural thrombus complicating anterior myocardial infarction: a meta-analysis. J Am Coll Cardiol 1993; 22:1004–9.
8. Prrillo IE. Heart disease and the eosinophil. N Engl J Med 1990; 323:1560–1.
9. Yamamoto H, Nakatani S, Hashimura K. Löffler’s endomyocarditis. Heart 2004 (in press).
10. Karalis DG, Chandrasekaran K, Victor MF, Ross JJ, Mintz GS. Recognition and embolic potential of intraaortic atherosclerotic debris. J Am Coll Cardiol 1991; 17:73–8.
11. Tunick PA, Rosenzweig BP, Katz ES, Freedberg RS, Perez JL, Kronzon I. High risk for vascular events in patients with protruding aortic atheromas: a prospective study. J Am Coll Cardiol 1994; 23:1085–90.
12. Tunick PA, Perez JL, Kronzon I. Protruding atheromas in the thoracic aorta and systemic embolization. Ann Intern Med 1991; 115:423–7.
13. Amarenco P, Cohen A, Tzourio C, et al. Atherosclerotic disease of the aortic arch and the risk of ischemic stroke. N Engl J Med 1994; 331:1474–9.
14. Sanfilippo AJ, Picard MH, Newell JB, et al. Echocardiographic assessment of patients with infectious endocarditis: prediction of risk for complications. J Am Coll Cardiol 1991; 18:1191–9.
15. Di Salvo G, Habib G, Pergola V, et al. Echocardiography predicts embolic events in infective endocarditis. J Am Coll Cardiol 2001; 37:1069–76.
16. Tazelaar HD, Locke TJ, McGregir CGA. Pathology of surgically excised primary cardiac tumors. Mayo Clin Proc 1992; 67:957–65.
17. Ha JW, Kang WC, Chung N, et al. Echocardiographic and morphologic characteristics of left atrial myxoma and their relation to systemic embolism. Am J Cardiol 1999; 83:1579–82.

18. Topol EJ, Biern RO, Reits BA. Cardiac papillary fibroelastoma and stroke. Echocardiographic diagnosis and guide to excision. Am J Med 1986; 80:129–32.
19. Cabanes L, Mas JL, Cohen A, et al. Atrial septal aneurysm and patent foramen ovale as risk factors for cryptogenic stroke in patients less than 55 years of age. A study using transesophageal echocardiography. Stroke 1993; 24:1865–73.
20. Lechat P, Mas JL, Lascault G, et al. Prevalence of patent foramen ovale in patients with stroke. N Engl J Med 1988; 318:1148–52.
21. Homma S, Sacco RL, Di Tullio MR, Sciacca RR, Mohr JP. PFO in cryptogenic stroke study (PICSS) investigators. Effect of medical treatment in stroke patients with patent foramen ovale: patent foramen ovale in cryptogenic stroke study. Circulation 2002; 105:2625–31.
22. Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. Mayo Clin Proc 1984; 59:17.
23. Schuchlenz HW, Weihs W, Horner S, Quehenberger F. The association between the diameter of a patent foramen ovale and the risk of embolic cerebrovascular events. Am J Med 2000; 109:456–62.
24. Lynch JJ, Schuchard GH, Gross CM, Wann LS. Prevalence of right-to-left atrial shunting in a healthy population: detection by Valsalva maneuver contrast echocardiography. Am J Cardiol 1984; 53:1478–80.
25. Hanley PC, Tajik AJ, Hynes JK, et al. Diagnosis and classification of atrial septum aneurysm by two-dimensional echocardiography: report of 80 consecutive cases. J Am Coll Cardiol 1985; 6:1370–82.
26. Schneider B, Hanrath P, Vogel P, Meinertz T. Improved morphologic characterization of atrial septal aneurysm by transesophageal echocardiography: relation to cerebrovascular events. J Am Coll Cardiol 1990; 16:1000–9.
27. Mugge A, Daniel WG, Angermann C, et al. Atrial septal aneurysm in adult patients. A multicenter study using transthoracic and transesophageal echocardiography. Circulation 1995; 91:2785–92.

8
Echocardiography in acute pulmonary embolism

Piotr Pruszczyk and Adam Torbicki

Key points

- Echocardiographic signs of right ventricular overload are indirect signs of acute pulmonary embolism (PE) and could result from other causes. However, these signs in patients with PE are associated with increased in-hospital risk, even in normotensive patients.
- McConnell's sign and the '60/60 sign' are more specific for acute PE and in some situations can justify an aggressive treatment approach.
- Transesophageal echocardiography (TEE) can be used for urgent bedside confirmation of significant PE (in about 80% of cases), even in intubated patients; however, due to its topographic limitations, it cannot rule out PE.
- Detection of right heart thrombi confirms clinically suspected PE.
- Detection of deep vein thromobosis by ultrasonography in a patient with clinically suspected acute PE supports the diagnosis and justifies anticoagulation.

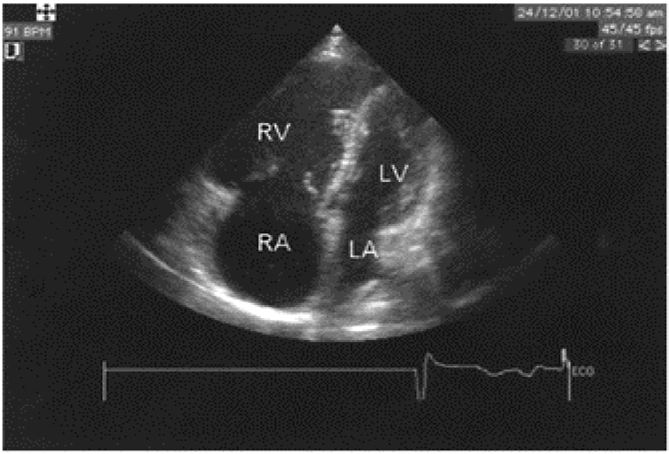
Acute pulmonary embolism (PE) remains one of the major causes of in-hospital mortality. Its prompt and accurate diagnosis is especially important in hemodynamically unstable patients, because they may require urgent thrombolysis or surgery. The noninvasive character of echocardiography and its high bedside availability are particularly useful in an emergency setting, and increasingly appreciated in suspected acute PE. Recent large-scale registry showed that, in the most hemodynamically compromised patients with suspected acute PE, routine diagnostic procedures, such as angiography, lung scintigraphy, and spiral computed tomography (sCT), were performed less frequently, and the diagnosis of PE was mainly based on clinical evaluation and echocardiography.[1] Moreover, in a retrospective study of 1246 cardiac arrest victims, echocardiography was the most commonly used imaging method, revealing the diagnosis in 24 out of 60 (40%) patients suffering from acute PE.[2] This chapter provides information which should help to optimize the use of echocardiography in emergency assessment of patients with suspected or confirmed acute PE.

Transthoracic echocardiography (TTE) in suspected acute PE

The hemodynamic consequences of acute PE depend not only on the degree of thrombotic load in pulmonary arteries, but also on individual cardiopulmonary reserve.

Therefore, in a young patient without pre-existing cardiopulmonary disorders, even total occlusion of one pulmonary artery may not alter right ventricular (RV) morphology and function, while in subjects with low cardiopulmonary reserve smaller pulmonary emboli can result in acute cor pulmonale with systemic hypotension or even shock. However, it can be reasonably expected that acute PE will cause acute RV pressure overload when obstruction of pulmonary vascular bed exceeds 30–50%[3] (Figure 8.1). The presence of RV strain can be diagnosed by measuring the peak velocity of the jet of tricuspid valve regurgitation. In addition, assessment of the pulmonary ejection pattern can reveal abnormalities highly suggestive of proximal obstruction of pulmonary arteries. Acutely increased systolic and filling pressures result in the dilation of right ventricle, right atrium, and inferior vena cava.[4–8] The intraventricular septum may be flattened and bulge into the left ventricle.[9] Thus, in an acute setting, with the pericardium limiting cardiac enlargement, the left ventricle becomes compressed and distorted by the expanding right ventricle. All those signs are suggestive of, but not fully diagnostic for, PE. Several studies assessed the sensitivity and specificity of the diagnosis of PE by various sets of echocardiographic criteria reflecting RV overload (Table 8.1).

According to the French authors who studied 132 patients with suspected acute PE, the combined criteria, including tricuspid regurgitant flow peak velocity greater than 2.5 m/s and a right over left ventricular diameter ratio greater than 0.5, as assessed with M-mode echocardiography, would permit the diagnosis of PE with sensitivity of 93%, and specificity of 81%.[10] However, this study excluded patients with a history of cardiac or pulmonary diseases, in whom signs of RV overload are often present and might adversely affect the specificity of the suggested echocardiographic criteria. Interestingly, the Italian group studying 117 consecutive, nonselected patients with suspected PE reported an even higher specificity (87%) but lower sensitivity (51%) of echocardiographic diagnosis of PE.[11] When integrated clinical, echocardiographic, and venous ultrasonographic evaluation was performed, overall sensitivity increased to 89% but specificity fell to 74%, which was lower than with echocardiographic evaluation alone. In addition, the echocardiographic criteria for diagnosis of acute PE suggested by this study were rather complex. At least one



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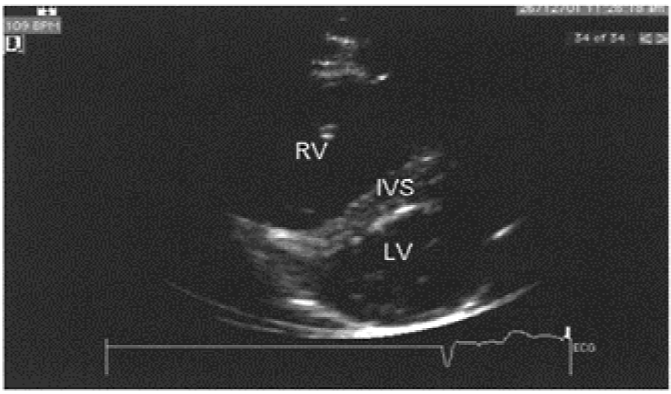

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Figure 8.1 (A) Apical four-chamber view. Enlarged right ventricle (RV) dominating left ventricle (LV). At real-time scanning, hypokinesis of middle segment of RV free wall was present with preserved contractility of its apical segment (McConnell's sign; see text). RA: right atrium; LA: left atrium. (B) Flattened intraventricular septum (IVS) bulging into left ventricle present in short-axis parasternal view.

Table 8.1 Major studies evaluating diagnostic value of indirect echocardiographic pulmonary embolism

Source	*n*	*Screened population*	*Echocardiographic criteria used*	*Sens*	*Spec*	*PPV*	*NPV*
Nazeyrollas et al[10]	132	Out-patients, no known previous serious cardio-respiratory disease	RV/LVEDD>0.5 (parasternal M-mode echo) TI jet velocity>2.5 m/s	93%	81%	78%	93%
Grifoni et al[11]	117	Consecutive patients seen at emergency department	One or more of four signs: 1. Right heart thrombus 2. RV >30 mm parasternal view or RV/LVED > 1.3 3. Systolic flattening of interventricular septum 4. AcT or <90 ms or TIPG >30mmHg but no RV hypertrophy	51%	87%	82%	60%
Perrier et al[12]	50	Consecutive patients, mostly from emergency ward	RV dilation 'by visual inspection' on 2-D echo and TI jet velocity >2.6 m/s	67%	94%	86%	83%
McConnell et al[17]	85	Hospitalized patients with RV dysfunction	Hypokinetic RV free wall but normo/hyperkinetic RV apex	77%	94%	71%	96%
Torbicki et al[19]	86	Hospitalized patients with precapillary pulmonary hypertension	AcT <60 ms with TIPG <60 mmHg	48%	98%	n.a.	n.a.
Miniati et al[13]	110	Consecutive patients with clinically suspected APE	Two of the following: signs: 1. RV hypokinesis, 2. RV diameter>27 mm, long parasternal 3. TI velocity>2.7 m/s	56%	90%		

RV, right ventricle; LV, left ventricle; EDD, end diastolic dimension; TI, tricuspid insufficiency; PG, pressure gradient; AcT, acceleration time of right ventricular ejection flow velocity curve; PE, pulmonary embolism; pts, patients; n.a., not assessed; sens, sensitivity; spec, specificity; PPV, positive predictor value; NPV, negative predictive value.

out of four signs was required (Table 8.1) in the absence of increased RV wall thickness. If RV wall thickness exceeded 6 mm, it was considered indicative of chronic pulmonary hypertension rather than acute PE.

A simpler echocardiographic approach was adopted in a recent Swiss single-center study,[12] including 50 consecutive patients with clinically suspected PE. In all cases, final diagnosis was reached by a sequential noninvasive strategy followed by pulmonary

angiography, wherever appropriate. PE was confirmed in 18 of 50 patients (36%). RV dilation on two-dimensional (2-D) echocardiography associated with a tricuspid regurgitation velocity of at least 2.7 m/s, was present in 12 out of 18 (67%) patients with confirmed and in 2 out of 32 (6.3%) patients with excluded PE. In this relatively small study group, echocardiographic criteria had a high specificity of 94%. When combined with high clinical probability of PE, those criteria seemed useful in selecting patients in whom prompt initiation of treatment was justified. Another Italian group showed that the clinical impact of echocardiographic signs of RV overload for the diagnosis of acute PE was highly influenced by the clinical probability of this disease.[13] Coexistence of at least two out of the three following signs, RV hypokinesis, RV enlargement, and systolic velocity of tricuspid valve regurgitation of >2.5 m/s, was analyzed. Thus, for pretest clinical probability of 10%, 50%, or 90%, post-test probability of acute PE was 38%, 85%, or 98%, respectively.

The assessment of the diameter of inferior vena cava during the respiratory cycle can also be useful in diagnosis and assessment of acute PE. In contrast to chronic pulmonary hypertension, the decreased collapsibility index of the inferior vena cava (IVCCI) is almost a rule in acute pulmonary hypertension such as that due to an episode of PE. When defined as inspiratory change in the inferior vena cava diameter of less than 40% of its

Table 8.2 Echocardiographic signs useful in the differentiation of causes of enlarged right ventricle

	Maximal tricuspid peak systolic gradient	*Acceleration time of RV ejection*	*Inferior vena cava*	*Others*
Acute RV overload in APE	30–60 mmHg	<80 ms, frequently <60 ms with midsystolic 'notch'	>20 mm, noncollapsing at inspiration	Hypokinesis of RV free wall, preserved contractility of its apical segment
CTEPH	Frequently >60 mmHg	<80 ms, frequently <60 ms with midsystolic 'notch'	>20 mm, noncollapsing at inspiration	Signs of RV hypertrophy; diastolic thickness of RV free wall >6 mm
Chronic cor pulmonae	Usually <80 mmHg	<80 ms infrequently with notch	>20 mm, collapsing at inspiration	Signs of RV hypertrophy; diastolic thickness of RV free wall >6 mm
RV infarction	Mostly<30 mmHg	>100ms	>20mm, noncollapsing at inspiration	Coexisting regional systolic wall motion abnormalities at inferior or posterior LV wall
Isolated tricuspid valve	Mostly<30 mmHg	>100ms	>20mm, noncollapsing at	Preserved contractility of both

regurgitation			inspiration	ventricles
LV dysfunction, mitral valve disease	Mostly<60 mmHg but may be higher	<100 ms, rarely midsystolic ‘notch’	>20 mm, noncollapsing at inspiration	Advanced LV systolic dysfunction or mitral valve pathology
Intracardiac shunts (early stage)	Mostly<60 mmHg	Normal or only slightly shortened<100 ms, signs of increased pulmonary cardiac output	Frequently normal	Signs of intracardiac shunts
Eisenmenger’s syndrome	Mostly >60 mmHg	Relatively less shortened than in other pathologies with similar level of PASP	Frequently normal	RV hypertrophy; intracardiac shunt usually with low pressure gradients

RV, right ventricle; LV, left ventricle; CTEPH, chronic thromboembolic pulmonary hypertension; PASP, pulmonary artery systolic pressure.

maximum expiratory value, decreased IVCCI was reported in 82% of 60 patients with PE presenting with RV dilation.[14,15] Interestingly, the collapsibility index was also the first echocardiographic sign to improve with treatment, correlating with normalization of right atrial pressure.

It should be underlined that RV strain is only an indirect sign of acute PE and may be caused by various acute cardiopulmonary conditions, including acute respiratory distress syndrome. In fact, Jardin et al found RV overload in 17/196 (8.7%) patients with adult respiratory distress syndrome (ARDS).[4] RV myocardial infarction can also lead to RV enlargement, dilation of tricuspid annulus, and even severe tricuspid regurgitation. Distension of inferior vena cava without respiratory changes is frequently present in such patients. However, RV infarction often accompanies myocardial infarction of the left ventricular inferior wall. Therefore, echocardiography usually reveals abnormalities of regional systolic function at the left ventricular inferior wall. Moreover, the pulmonary ejection pattern is not disturbed and tricuspid valve peak systolic gradient is not increased. The echocardiographic data summarized in Table 8.2 may be helpful in the differentiation of RV enlargement.

While signs of RV pressure overload in an appropriate clinical setting are highly suggestive of acute PE, they are not fully reliable. It is very important to remember that false-positive diagnosis may lead to an unnecessary thrombolysis with all its inherent risks. Since MAPPET registry showed that almost one-third of patients with acute PE who received thrombolysis had relative contraindications for such treatment,[1] and the ICOPER study reported a very high incidence of major bleeding (21%), including intracranial hemorrhage (3%), in

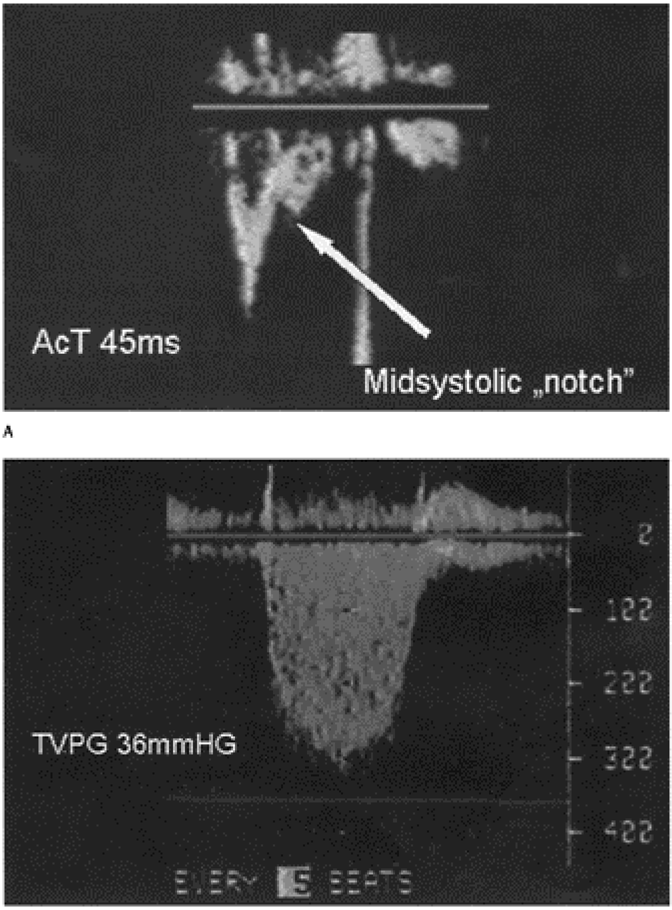

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Figure 8.2 Disturbed pulmonary ejection assessed with PW Doppler in right ventricular outflow tract. (A) Markedly shortened acceleration time (AcT) of 45 ms with midsystolic notch (notch) in a patient with massive acute PE but only (B) moderately elevated tricuspid valve peak systolic gradient (below 60 mmHg). An example of the '60/60 sign' (see text).

thrombolysed patients,[16] definitive confirmation of acute PE, whenever possible, is especially important.

Recently, more specific echocardiographic signs of acute PE have been defined. Hypokinesis of the RV free wall with preserved contractility of its apical segment was suggested by McConnell et al as useful to distinguish between acute pressure overload caused by acute PE and other diseases leading to RV strain (Figure 8.1).[17] McConnell's sign showed a specificity of 94% with sensitivity of 77% in the retrospective analysis of 85 patients. Although the pathophysiology of this phenomenon has not yet been clearly defined, successful treatment of acute PE led to the recovery of RV regional function.[18]

Doppler echocardiography can also provide signs that appear to be more specifically linked to acute PE. Characteristic disturbances of the flow velocity curve of RV ejection were observed in patients with hemodynamically significant acute PE. Because pressure waves reflected from centrally located pulmonary arterial masses interfere with pulmonary ejection, this results in markedly shortened acceleration time (AcT) and midsystolic deceleration (midsystolic notch) (Figure 8.2). Acceleration time can be very short in patients with central acute PE, even in those with only slightly increased RV systolic pressure. Coexistence of acceleration time of pulmonary ejection below 60 ms measured in RV outflow tract and tricuspid valve

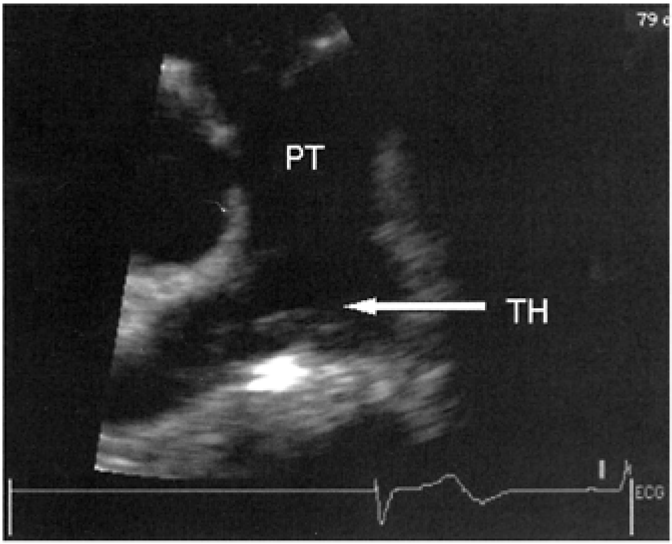


Figure 8.3 Saddle thromboembolus (TH) lodged in main pulmonary trunk (PT) and extending into both main left and main right pulmonary arteries, as visible from modified short-axis parasternal view. (Reproduced from Echocardiography. Hoffman P, Kasprzak JD (eds). Gdansk: ViaMedica, 2004.)

peak systolic gradient of less than 60 mmHg (‘60/60 sign’) is highly suggestive of acute PE. In a retrospective observation of 86 patients with various causes of pulmonary hypertension, this 60/60 sign was 94% specific, with a fair sensitivity of 48% for the diagnosis of acute PE.[19] In a prospective study of 100 consecutive patients with clinically suspected PE, the clinical value of both McConnell’s sign and the 60/60 sign was assessed.[20] Importantly, this group comprised unselected patients and included subjects with various cardiopulmonary diseases. Acute PE was confirmed by reference tests in 67/100 patients. Sensitivity and specificity were 25% and 94% for the 60/60 sign, and 19% and 100% for McConnell’s sign. When combined, the two signs were 94% specific and 36% sensitive in diagnosing acute PE. In our opinion, if McConnell’s sign and/or the 60/60 sign are detected, even in a patient without clinical suspicion of acute PE and with a potential explanation of hemodynamic instability or dyspnea, acute PE still should be strongly suspected, and suitable diagnostic measures should be undertaken. The major limitation of both McConnell’s sign and the 60/60 sign is limited sensitivity. Therefore, their absence or even the lack of any echocardiographic signs of RV overload cannot exclude acute PE. However, hemodynamically stable patients with no echocardiographic RV dysfunction constitute a group of nonmassive acute PE, a ‘benign’ form of the disease, with good prognosis.

Visualization of thrombi

PE can be confirmed by direct visualization of thromboemboli inside a pulmonary artery. It is generally accepted that [c]in transit’ thrombi found in right heart chambers also prove acute PE. Moreover, recently, it was suggested that detection of thrombi in the peripheral venous system in patients with clinically suspected acute PE discloses a potential source of emboli and justifies treatment. Documentation of venous thrombi may be especially helpful for therapeutic decisions in patients in whom echocardiographic signs of RV overload have suggested but not established the diagnosis of PE.

Pulmonary artery thrombi

Transthoracic echocardiography (TTE) only infrequently allows direct visualization of pulmonary artery thrombi. However, the pulmonary trunk and the initial parts of pulmonary arteries can be assessed from the subcostal approach or from the modified short-axis parasternal view (Figure 8.3). The suprasternal window enables visualization of the right pulmonary artery, and it was reported to be useful for thrombi detection.[5] Although we encourage the use of all these approaches in a patient with

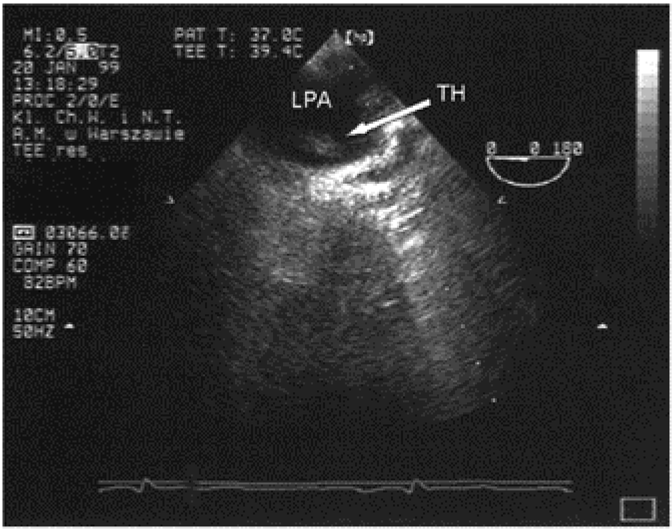


Figure 8.4 Pulmonary artery thrombi (TH) visualized by TEE. Mobile thrombus detected in the distal part of the left main pulmonary artery (LPA).

suspected acute PE, the chance of unequivocal visualization of pulmonary arterial thrombus is rather low.

Transesophageal echocardiography (TEE) improved visualization not only of heart structures but also of great vessels including proximal parts of pulmonary arteries. Since the first report by Nixdorff et al, who detected pulmonary artery thrombus during TEE,[21] many case reports and short series have indicated the clinical value of TEE in the prompt confirmation of acute PE. More systematic studies were performed to assess the clinical value of TEE in suspected acute PE.[22–25] High specificity of TEE diagnosis can be expected only when unequivocally visualized intrapulmonary masses with distinct borders, different in echodensity from the vascular wall, are reported as thromboemboli (Figure 8.4). The echocardiographic diagnosis of thrombi should always be done with the understanding that the result of TEE can serve as a justification of aggressive treatment, including thrombolysis or even embolectomy. Therefore, at the beginning of the learning curve, special care should be taken not to overdiagnose acute PE.[26]

The limited sensitivity of TEE for acute PE results from an imaging window restricted to central pulmonary arteries. In the early studies, the left pulmonary artery was considered inaccessible to TEE evaluation, because it was shielded from the ultrasound beam by the left main bronchus.[22,25,27] Wittlich et al studied 60 patients with confirmed PE and signs of RV overload, finding 32 thrombi in the right, and only 6 in the left pulmonary artery.[25] Modification of the scanning techniques used in our laboratories extended examination also to the left pulmonary artery in the majority of cases.[28]

Standard TEE in a patient with suspected acute PE usually begins with the evaluation of the main pulmonary artery. Then the right pulmonary artery is investigated both in the transverse and longitudinal planes up to the initial parts of lobar arteries, which is possible with appropriate clockwise rotation and slight advancing of the probe. An immobile, side-lobe artifact, in the middle portion of the right pulmonary artery located close to the esophagus, is present in some patients. It may be misinterpreted as thrombus, and color Doppler may be needed to avoid false-positive diagnosis. Counterclockwise rotation of the transducer allows evaluation of the left pulmonary artery. Here, the echocardiographic continuity of the artery is often lost, due to the interposition of the left main bronchus. However, with further transducer rotation, the distal part of the left pulmonary artery can be identified in approximately 80% of patients. Its position with respect to the descending aorta, the characteristic branching of the left upper lobe artery, and the appearance of air microbubbles after saline injection into the antecubital vein help to define the left pulmonary artery. Efforts are made to visualize both pulmonary arteries as distally as possible, including the initial parts of lobar arteries where many of the thromboemboli tend to lodge. If the examination is tolerated well, the intra-atrial septum is searched for the patent foramen ovale (PFO), and venous return to the left atrium from each of the four pulmonary veins is assessed.

The higher the degree of obstruction of pulmonary arteries by thromboemboli, the higher is the probability of their visualization by TEE in central pulmonary arteries. Therefore, the selection criteria of patients for TEE strongly influence its sensitivity. In our laboratories, only patients with RV overload at TTE undergo tranesophageal examination to detect thrombi. In a group of 113 patients with clinical suspicion of PE presenting RV overload, TEE reached sensitivity of 80% with specificity of 97.4%.[29] However, sensitivity would be probably much lower in an unselected group of patients, which would include many patients with PE limited to smaller and more distal thrombi.

Since TEE has topographic limitations, a question arises regarding its role in case of the availability of alternative imaging methods. Direct comparison of the diagnostic power of TEE and sCT was prospectively performed in 49 consecutive patients with unexplained RV overload. In this preselected group with high embolic load in the proximal pulmonary arteries, the sensitivity of TEE was only slightly lower than that of sCT (79% and 97.5%, respectively). However, TEE was at least as specific as sCT (100% and 90%) and could be performed at the bedside.[30]

The experience of the echocardiographic team may also influence the diagnostic value of TEE. Therefore, routine evaluation of pulmonary arteries in patients without suspicion of PE might improve the diagnostic impact of TEE.

Once intraluminal masses are detected in the pulmonary arteries, TEE can help to assess their morphology and origin. Mobile 'snake-like' structures suggest acute PE, while immobile perimural masses with increased echodensity may suggest chronic thromboembolic pulmonary hypertension.[24,25,31] In rare cases, intraluminal masses may not be of embolic origin, and may represent *in situ* thrombosis developing in some patients with longlasting pulmonary hypertension[32,33] or they may represent primary neoplasms of pulmonary arteries, such as leiomyosarcoma.[34] Interestingly, other imaging methods such as sCT, scintigraphy, or pulmonary angiography cannot differentiate these findings. However, intraluminal masses in patients with shock, hypotension, or acute

dyspnea, especially mobile, detected in the proximal parts of pulmonary arteries, should be regarded as thromboemboli and should have immediate and appropriate treatment.

Apart from the direct visualization of thrombotic material in pulmonary arteries, TEE may provide additional data potentially useful in the diagnosis and prognosis of PE. Asymmetry of venous blood inflow into the left atrium can be observed in some patients with PE and may correspond with the region of significantly reduced perfusion observed at lung scintigraphy. Assessment of the intra-atrial septum allows detection of the PFO, the presence of which was reported to indicate a worse prognosis.[35]

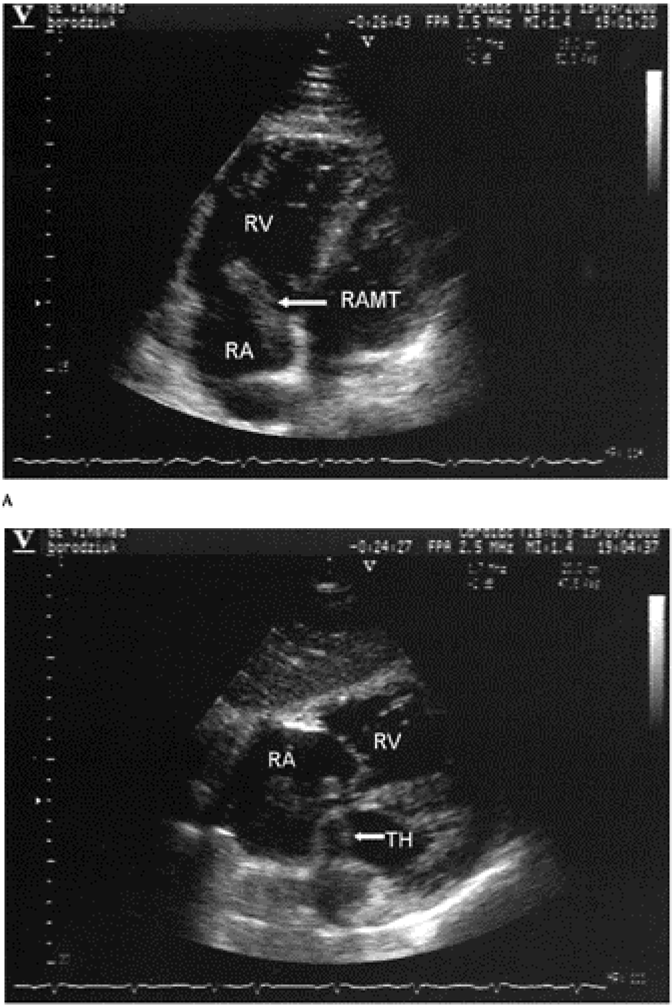
In summary, TEE performed in suspected massive acute PE, in our opinion, is a safe method, which can be easily performed also in mechanically ventilated patients. Pulmonary arteries can be evaluated within a few minutes, and the procedure can be stopped after the first unequivocal thrombus has been detected. Interestingly, the diagnosis of PE was established by TEE even in patients with shock[36] or during cardiopulmonary resuscitation.[37,38]

Clinical implications of TEE

TEE of central pulmonary arteries is a reliable, safe method of definitive confirmation, though not exclusion, of hemodynamically significant PE. As a prompt, bedside examination, TEE can rapidly detect clots in 80% of cases of PE presenting with RV overload. If we consider also the possibility of bedside evaluation, TEE may be the first choice method even if sCT is available, especially in hemodynamically compromised patients who may require urgent thrombolysis or surgery. Direct visualization of intrapulmonary thrombi may be of special importance in patients with suspected massive acute PE and coexisting relative contraindication for thrombolysis. However, visualization of a high degree of thrombotic obstruction of pulmonary arteries or saddle acute PE39,[40] per se is not an indication for aggressive treatment, and therapy selection should be based mostly on the clinical status of the patient. Interestingly, it has been suggested that the morphology of pulmonary artery thromboemboli assessed during TEE may have an impact on the effect of thrombolysis and short-term outcome. Patients with immobile central pulmonary thromboemboli have a worse short-term outcome than those with mobile central thromboemboli.[31]

Right heart thrombi

Echocardiography may detect thrombi in the right heart chambers. Usually, they are found in the right atrium and frequently present as highly mobile structures sometimes prolapsing during diastole into the right ventricle (Figure 8.5).[41–43] Their snake-like appearance and coexistence with deep vein thrombosis often suggest venous origin. In most cases, mobile right heart thrombi are highly unstable and



A

B

Figure 8.5 (A) Right atrium (RA) mobile thrombus (RAMT) (arrow) in diastole prolapsing into the right ventricle (RV). (B) Subcostal approach detects penetration of thrombus (TH) via patent foramen ovale into the left atrium. (Reproduced from Echocardiography. Hoffman P, Kasprzak JD (eds). Gdansk: ViaMedica, 2004.)

tend to embolize to pulmonary arteries. Immediate aggressive treatment is indicated, but the optimal mode of therapy remains controversial. Recently, thrombolytic treatment seems to be preferred to surgery, as can be judged from published case reports. However, thrombolysis should be avoided because of the risk of systemic embolism and embolic stroke, if a right atrial thrombus extends through the PFO into the left atrium (Figure 8.5b). Such left-sided protrusion of a thrombus is not always easy to exclude without recurring to TEE. Unfortunately, dislodgment of right heart thrombi with resulting fatal PE has been reported during introduction of an esophageal probe. This was attributed to shifts in intrathoracic pressure induced by the gag reflex. Therefore, either very efficient sedation should be provided during TEE, or surgery should be offered to the patient in case of doubt regarding the position of the right heart thrombus with respect to the intra-atrial septum. Surgical treatment is attractive also when the clinical context and/or appearance of the right heart mass on echocardiography may suggest its nonthrombotic origin. The possibility of myxoma or extracardiac tumor extending into the right atrium via the inferior or superior vena cava should always be considered before the final decision of optimal therapy is taken. Unfortunately, in clinical practice, decisions regarding treatment of mobile right heart masses are among the most difficult to make. However, recent analysis of international registry showed that among patients with acute PE right heart thrombi are usually found in those more hemodynamically compromised. Moreover, it was indicated that such thrombi are markers of worse prognosis in initially apparently stable patients treated with heparin alone, suggesting the need for more aggressive therapy.[44] With the increasing availability of TTE, most of the cases are diagnosed in centers without immediate access to cardiac surgery. As the transport of a patient with floating right heart mass to a referral center for TEE and/or embolectomy clearly increases the risk of its dislodgment, every effort should be made to begin the treatment without moving the patient from the hospital which made the diagnosis. Apart from echocardiographic findings, the clinical condition of the patient is crucial for decision making, and in the presence of life-threatening hypotension or shock, thrombolysis should not be delayed if surgery is not immediately available. Despite the high risk of systemic embolization, cases with good outcome were also reported among patients with impending paradoxical embolism. On the other hand, a small right heart thrombus in a patient with only mild cardiorespiratory compromise probably does not significantly alter the prognosis. However, whether heparin treatment alone in such cases is sufficient remains unclear.

'Integrated ultrasound' approach

PE is one of the clinical presentations of venous thromboembolism (VTE). Deep venous thrombosis (DVT) of low extremities assessed by contrast venography is present in 70% of cases of acute PE. Clinical signs of DVT in patient with acute dyspnea increase the probability of acute PE. However, approximately 50% of cases of DVT are clinically silent. This indicates the clinical need for an objective, preferably noninvasive, and widely available method to detect venous thrombosis. Venous ultrasound (VUS) has a well-established value in the diagnosis of patients with suspected DVT.[45] Although VUS can detect venous thrombi less frequently than contrast venography—in approximately 50% of patients with acute PE—it is highly specific, potentially usable at the bedside, and

totally noninvasive.[46] Furthermore, a simplified protocol limited to groin and popliteal vessels may be performed rapidly and is easy to learn for an experienced echocardiographer. Detection of DVT in a patient with suspected acute PE virtually confirms the diagnosis and fully justifies anticoagulation. This is especially important when no immediate access to reference imaging methods is available. Confirmation of DVT with compression ultrasound is particularly rewarding in patients who have RV pressure overload but also a potential alternative cause, such as chronic obstructive pulmonary disease (COPD). Moreover, in an unstable patient with RV pressure overload, documentation of a thrombus in proximal veins makes the decision to start thrombolytic treatment without further delay much easier.

Echocardiography, especially TEE, allows direct visualization of thromboemboli in the proximal parts of pulmonary arteries, while TTE can detect ‘in transist’ thrombi on their way to pulmonary circulation. In addition, ultrasound scanning of peripheral venous system can find sources of emboli and confirm venous thromboembolic disease. Therefore, the extension of standard TTE to TEE, and to peripheral venous scanning increases the diagnostic impact of ultrasound examination. This ‘integrated ultrasound approach’ can be performed by a single echocardiographer using a single machine equipped with three probes, transthoracic, transesophageal, and vascular, at the bedside of an unstable patient in the intensive care unit, or even in the operating room prior to emergency embolectomy.

Definitive confirmation of PE with TEE can be expected in 80% of patients with signs of RV overload. Compression venous ultrasound, however, is noninvasive, may be performed regardless of recently digested meals, and reliably confirms proximal venous thrombosis even when performed with classic cardiologic phased-array, 3.5-MHz probes.

In our opinion ‘integrated ultrasound’ represents major progress in the emergency diagnosis and management of suspected acute PE. Clearly, this approach is useful for confirming, but not excluding, venous thromboembolic disease. If integrated ultrasound fails to document intravascular/intracardiac thrombi, further diagnostic procedures are warranted.

Suggested diagnostic strategy for severely compromised patients

In patients with unexplained hypotension or shock emergency, echocardiography is indicated (Figure 8.6). Normal RV morphology and function make massive acute PE highly unlikely; hence, alternative causes of hemodynamic instability should be considered. In patients with RV strain, massive acute PE should be strongly suspected.

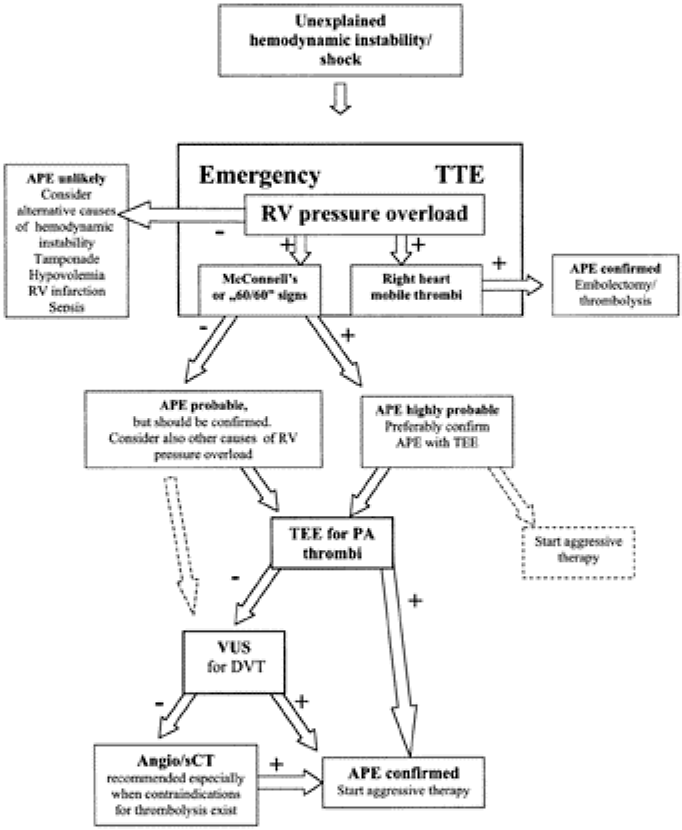


Figure 8.6 Diagnostic strategy in patient with unexplained shock or hypotension. APE, acute pulmonary embolism; RV, right ventricular; DVT, deep venous thrombosis; PA, pulmonary artery; TEE, transesophageal echocardiography; TTE, transthoracic echocardiography; VUS, venous ultrasound.

However, signs of RV overload may result from other causes such as COPD, pneumonia, and ARDS. More specific echocardiographic signs, such as McConnell's sign and the 60/60 sign, make the diagnosis highly probable although still not definite. Although aggressive therapy can be justified in life-threatening situations, further diagnostic work-up is suggested if possible. Recently, it was reported that patients with a high clinical pretest probability of acute PE and a shock index (heart rate divided by systolic blood pressure) of ≥1 presenting RV dysfunction at TTE were successfully urgently

thrombolysed without any further diagnostic work-up.[47] In infrequent cases, TTE solves the problem by visualization of thromboemboli in right heart chambers or initial parts of pulmonary arteries. Otherwise, bedside confirmation of intrapulmonary thrombi with TEE is an option, especially useful in unstable patients with relative contraindications for thrombolysis. Importantly, vascular ultrasonography performed according to a simplified protocol focusing on the groin and popliteal region may promptly confirm venous thromboembolism, making the decision to start the treatment much easier. In most cases, however, after stabilization of the patient, either lung scan, sCT, or angiography should be performed to confirm acute PE.

Alternative diagnosis

Echocardiography can detect alternatives to acute PE causes of acute hemodynamic instability, dyspnea, shock, or chest pain. It has a well-documented role in the diagnosis of acute left ventricular systolic dysfunction, cardiac tamoponade, aortic dissection, and infective endocarditis. Among 132 consecutive patients with clinically suspected PE, echocardiography was reported to reveal alternative causes in 42% of cases.[10] Moreover, echocardiography may unexpectedly find signs suggestive of acute PE in patients initially referred for other reasons. A typical case is that of ‘cryptogenic stroke’, which may in fact result from right to left shunting of blood containing venous clots originally in transit to pulmonary arteries but which wandered to the left atrium and systemic circulation through the PFO.

Echocardiography in confirmed acute PE

Echocardiography was found to be useful not only in the diagnosis of suspected acute PE, but also in the evaluation of prognosis in patients with proven acute PE. There is accumulating evidence that RV dysfunction indicates a worse prognosis. In-hospital mortality was reported to be significantly higher in patient with RV dysfunction, ranging from 12.6% to 15.9%, as compared to those with preserved RV function (0–8%).[16,48,49] A recent trial focusing on normotensive patients with acute PE and RV dysfunction reported slightly lower mortality (5%).[50] However, 6/65 (10%) patients with RV dysfunction developed shock despite heparin treatment, and three of them died. It should be underlined that echocardiographic criteria applied to the diagnosis of RV overload differ between published studies. This indicates the necessity of a prospective, multicenter study using uniform protocol to determine echocardiographic variables defining the degree of RV strain that could more precisely identify high-risk patients potentially requiring aggressive treatment. While subjectively diagnosed RV dysfunction in a normotensive patient with confirmed acute PE can be qualified as ‘submassive’ PE, according to the definition of the European Society of Cardiology (ESC),[51] no recommendations regarding thrombolytic treatment can be made for this category of patients at that time.

Other diagnostic parameters can help to distinguish further high-risk patients requiring special treatment among those with submassive acute PE. It was suggested that myocardial damage of the overloaded RV with its irreversible failure may precipitate

fatal outcome in acute PE. Increased plasma levels of cardiac troponins were reported in approximately 30% of patients with acute PE, especially those with clinically massive embolism, or with echocardiographic signs of RV overload.[52–54] Moreover, increased plasma troponin concentrations were found to be an independent risk factor for fatal outcome, and their evaluation was suggested to be useful in risk assessment.[53,54] Our study indicates that, among subjects with acute PE normotensive on admission, neither systemic systolic blood pressure nor dilation of the right ventricle distinguished survivors from nonsurvivors in-hospital. However, patients with detectable plasma levels of cardiac troponin T formed a high-risk group with in-hospital mortality of 25%.[55] We observed also that RV strain persisting for several hours, despite apparently effective treatment, may result in negative T waves developing in precordial ECG leads as late as 2–3 days after an acute episode. This deferred myocardial injury was often confirmed by delayed elevation of plasma cTnT. On the other hand, normotensive patients without RV dysfunction have a good prognosis, especially when no elevation of cardiac troponins is detected.[56] Recently, brain natriuretic peptides were reported to be elevated in patients with acute PE and RV overload.[57] Interestingly, NT-proBNP was found to reflect the degree of RV overload.[58] Moreover, low levels of BNP or NT-proBNP indicated good prognosis.[57]

Suggested management strategy of submassive acute PE

Although no formal recommendations can be made, patients in our institutions with submassive acute PE, defined according to the ESC, are monitored in the intensive care unit but initially receive heparin alone (Figure 8.7). ECG and plasma troponin level are determined at admission. Patients with signs of myocardial injury form the high-risk group and are considered for thrombolysis. However, the decision of aggressive therapy is strongly influenced by the assessment of individual risk of bleeding. Patients without elevated plasma cTnT continue on anticoagulation. If clinical deterioration, or even no improvement after several hours, is observed, repeated assessment of plasma cTnT is performed. Patients with elevated cTnT are considered at high risk, and thrombolytic therapy is once more considered. The remaining patients receive further anticoagulation with regular clinical assessment. A prospective trial is needed to assess the clinical value of this management strategy.

Patent foramen ovale (PFO)

The PFO is present in 20–30% of the general population, and is accepted as a risk factor for ischemic stroke or peripheral embolism. Elevated right atrial pressure in patients with RV dysfunction secondary to acute PE predisposes to right-to-left intracardial shunt, leading to pronounced hypoxemia. Moreover, paradoxical peripheral embolism may complicate acute PE. One study assessed the frequency and prognostic significance of the PFO in acute PE resulting in pulmonary hypertension and RV dilation.[35] The diagnosis was made if at least five microbubbles of contrast (5.5% oxypolygelatin solution) injected into a peripheral vein could be seen in the left heart chambers with delay not exceeding three cardiac cycles after opacification of the right atrium. Interestingly, in 48/137 (35%) of patients with thus diagnosed PFO, in-hospital mortality was

significantly higher than in the remaining 91 patients (33% vs 14%, P<0.015). Peripheral arterial emboli, as well as episodes of ischemic stroke, were more frequent in patients with right to left shunt, contributing to increased mortality. However, there was also a trend toward a higher rate of intracranial bleeding in patients with PFO (4.2% vs 1.1%). This could be due either to slightly more frequent use of thrombolytic treatment (46% vs 38%, P=0.47) or to possible previous paradoxical emboli resulting in subclinical ischemic foci in CNS, which in turn caused hemorrhage during thrombolysis, or both. Unfortunately, although the PFO at contrast echocardiography seems to be a marker of increased risk among patients suffering from PE, no evidence supporting the efficacy of specific modifications in standard management is available as yet.

Summary

Ultrasound examination, including standard TTE, extended to compression ultrasound for proximal deep vein thrombosis and to TEE in patients with RV pressure overload at TTE, can promptly confirm acute PE, or at least make the diagnosis highly probable in the majority of patients with suspected massive acute PE. Echocardiography can be performed in the emergency room, intensive care unit, or operating theater. Its bedside application makes echocardiography especially attractive in severely compromised patients, who should be diagnosed as quickly as possible, preferably without being transported. Moreover, echo can provide an alternative explanation of hemodynamic instability. However, it should always be remembered that RV strain at TTE is only an indirect sign of acute PE and confirmation is advisable, if possible. Normal RV function does not exclude acute PE, but, even in its presence, indicates excellent prognosis on treatment limited to heparin. In contrast, patients with RV overload are at higher risk of in-hospital death. They should be carefully monitored for signs of progressive RV myocardial injury and may require thrombolytic treatment even in the absence of shock or hypotension.

References

1. Kasper W, Konstantinides S, Geibel A, et al. Management strategies and determinants of outcome in acute major pulmonary embolism: results of a multicenter registry. J Am Col Cardiol 1997; 30:1165–71.
2. Kurkciyan I, Meron G, Sterz F, et al. Pulmonary embolism as a cause of cardiac arrest: presentation and outcome. Arch Intern Med 2000; 160:1529–35.
3. Wolfe MW, Lee RT, Feldstein ML, Parker JA, Come PC, Goldhaber SZ. Prognostic significance of right ventricular hypokinesis and perfusion lung scan defects in pulmonary embolism. Am Heart J 1994; 127:1371–5.
4. Jardin F, Dubourg O, Bourdarias JP. Echocardiographic pattern of acute cor pulmonale. Chest 1997; 111:209–17.
5. Kasper W, Meinertz T, Henkel B, et al. Echocardiographic findings in patients with proved pulmonary embolism. Am Heart J 1986; 112:1284–90.
6. Kasper W, Geibel A, Tiede N, et al. Distinguishing between acute and subacute massive pulmonary embolism by conventional and Doppler echocardiography. Br Heart J 1993; 70:352–6.

7. Torbicki A, Tramarin R, Morpurgo M. Role of echo/Doppler in the diagnosis of pulmonary embolism. Clin Cardiol 1992; 15:805–10.
8. Torbicki A. Echocardiography in pulmonary embolism. In: Morpurgo M (ed). Pulmonary embolism. New York: Marcel Dekker, 1994:153–74.
9. Jardin F, Dubourg O, Gueret P, Delorme G, Bourdarias JP. Quantitative two dimensional echocardiography in massive pulmonary embolism: emphasis on ventricular interdependence and leftward septal displacement. J Am Coll Cardiol 1987; 10:1201–6.
10. Nazeyrollas P, Metz D, Jolly D, et al. Use of transthoracic Doppler echocardiography combined with clinical and electrocardiographic data to predict acute pulmonary embolism. Eur Heart J 1996; 17:779–86.
11. Grifoni S, Olivotto I, Cecchini P, et al. Utility of an integrated clinical, echocardiographic, and venous ultrasonographic approach for triage of patients with suspected pulmonary embolism. Am J Cardiol 1998; 82:1230–5.
12. Perrier A, Tamm C, Unger PF, Lerch R, Sztajzel J. Diagnostic accuracy of Doppler-echocardiography in unselected patients with suspected pulmonary embolism. Int J Cardiol 1998; 65:101–9.
13. Miniati M, Monti S, Pratali L, et al. Value of transthoracic echocardiography in the diagnosis of pulmonary embolism: results of a prospective study in unselected patients. Am J Med 2001; 110:528–35.
14. Cheriex EC, Sreeram N, Eussen YF, Pieters FA, Wellens HJ. Cross sectional Doppler echocardiography as the initial technique for the diagnosis of acute pulmonary embolism. Br Heart J 1994; 72:52–7.
15. Simonson JS, Schiller NB. Sonospirometry: a new method for noninvasive estimation of mean right atrial pressure based on two dimensional echocardiographic measurements of the inferior vena cava during inspiration. J Am Cardiol 1988; 11:557–64.
16. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet 1999; 353:1386–9.
17. McConnell MV, Solomon SD, Rayan ME, Come PC, Goldhaber SZ, Lee RT. Regional right ventricular dysfunction detected by echocardiography in acute pulmonary embolism. Am J Cardiol 1996; 78:469–73.
18. Nass N, McConnell MV, Goldhaber SZ, Chyu S, Solomon SD. Recovery of regional right ventricular function after thrombolysis for pulmonary embolism. Am J Cardiol 1999; 83:804–6, A10.
19. Torbicki A, Kurzyna M, Ciurzynski M, et al. Proximal pulmonary emboli modify right ventricular ejection pattern. Eur Respir J 1999; 13:616–21.
20. Kurzyna M, Torbicki A, Pruszczyk P, et al. Disturbed right ventricular ejection pattern as a new Doppler echocardiographic sign of acute pulmonary embolism. Am J Cardiol 2002; 90:507–11.
21. Nixdorff U, Erbel R, Drexler M, Meyer J. Detection of thromboembolus of the right pulmonary artery by transesophageal two-dimensional echocardiography. Am J Cardiol 1988; 61:488–9.
22. Antakly-Hanon Y, Vieillard-Baron A, Qanadli SD, et al. The value of transesophageal echocardiography for the diagnosis of pulmonary embolism with acute pulmonary heart disease. Arch Mal Coeur Vaiss 1998; 91:843-8.
23. Fournier P, Augusseau Richard MP, Charbonnier B, Pettier JM, Pigale C, Pacouret G. Contribution of transesophageal echocardiography to the diagnosis of pulmonary embolism. Arch Mal Coeur Vaiss 1994; 87:459-65.
24. Pruszczyk P, Torbicki A, Kuch-Wocial A, Chlebus M, Miskiewicz ZC, Jedrusik P. Transoesophageal echocardiography for definitive diagnosis of haemodynamically significant pulmonary embolism. Eur Heart J 1995; 16:534-8.
25. Wittlich N, Erbel R, Eichler A, et al. Detection of central pulmonary artery thromboemboli by transesophageal echocardiography in patients with severe pulmonary embolism. J Am Soc Echocardiogr 1992; 5:515-24.

26. Torbicki A. Imaging venous thromboembolism with emphasis on ultrasound, chest CT, angiography and echocardiography. Thromb Haemost 1999; 82:907-12.
27. Vieillard-Baron A, Qanadli SD, Antakly Y, et al. Transesophageal echocardiography for the diagnosis of pulmonary embolism with acute cor pulmonale: a comparison with radiological procedures. Int Care Med 1998; 24:429-33.
28. Pruszczyk P, Torbicki A, Kuch Wocial A, Szulc M. Visualization of pulmonary arteries during transesophageal echocardiography. Clin Exp Cardiol 2001; 4:206-10.
29. Pruszczyk P, Torbicki A, Kuch Wocial A, Szulc M, Pacho R. Diagnostic value of transesophgeal echocardiography in suspected haemodynamically significant pulmonary embolism. Heart 2001; 85:628-34.
30. Pruszczyk P, Torbicki A, Pacho R, et al. Noninvasive diagnosis of suspected severe pulmonary embolism: transesophageal echocardiography vs spiral CT. Chest 1997; 112:722-8.
31. Podbregar M, Krivec B, Voga G. Impact of morphologic characteristics of central pulmonary thromboemboli in massive pulmonary embolism. Chest 2002; 122:973-9.
32. Moser KM, Fedullo PF, Finkbeiner WE, Golden J. Do patients with primary pulmonary hypertension develop extensive central thrombi? Circulation 1995; 91:741-5.
33. Russo A, De Luca M, Vigna C, et al. Central pulmonary artery lesions in chronic obstructive pulmonary disease: a transesophageal echocardiography study. Circulation 1999; 100:1808-15.
34. Parish J, Rosenow EC, Swensen SJ, Crotty TB. Pulmonary artery sarcoma. Chest 1996; 110:1480-8.
35. Konstantinides S, Geibel A, Kasper W, Olschewski M, Blumel L, Just H. Patent foramen ovale is an important predictor of adverse outcome in patients with major pulmonary embolism. Circulation 1998; 97:1946-51.
36. Krivec B, Voga G, Zuran I, et al. Diagnosis and treatment of shock due to massive pulmonary embolism: approach with transesophageal echocardiography and intrapulmonary thrombolysis. Chest 1997; 112:1310-16.
37. van der Wouw PA, Koster RW, Delemarre BJ, et al. Diagnostic accuracy of transesophageal echocardiography during cardiopulmonary resuscitation. J Am Col Cardiol 1997; 30:780-3.
38. Comess KA, DeRook FA, Russell ML, Tognazzi-Evans TA, Beach KW. The incidence of pulmonary embolism in unexplained sudden cardiac arrest with pulseless electrical activity. Am J Med 2000; 109:351-6.
39. Torbicki A, Pacho R, Jedrusik P, Pruszczyk P. Noninvasive diagnosis and treatment of a saddle pulmonary embolism. A case report in support of new trends in management of pulmonary embolism. Chest 1996; 109:1124-6.
40. Pruszczyk P, Pacho R, Ciurzynski M, et al. Short term clinical outcome of acute saddle pulmonary embolism. Heart 2003; 89:335-6.
41. Casazza F, Bongarzoni A, Centonze F, Morpurgo M. Prevalence and prognostic significance of right-sided cardiac mobile thrombi in acute massive pulmonary embolism. Am J Cardiol 1997; 79:1433-5.
42. Chapoutot L, Nazeyrollas P, Metz D, et al. Floating right heart thrombi and pulmonary embolism: diagnosis, outcome and therapeutic management. Cardiology 1996; 87:169-74.
43. Chartier L, Bera J, Delomez M, et al. Free-floating thrombi in the right heart: diagnosis, management, and prognostic indexes in 38 consecutive patients. Circulation 1999; 99:2779-83.
44. Torbicki A, Galie N, Covezzoli A, Rossi E, De Rosa M, Goldhaber SZ. Right heart thrombi in pulmonary embolism: results from the International Cooperative Pulmonary Embolism Registry. J Am Coll Cardiol 2003; 41:2245-51.
45. Kearon C, Ginsberg JS, Hirsh J. The role of venous ultrasonography in the diagnosis of suspected deep venous thrombosis and pulmonary embolism. Ann Intern Med 1998; 129:1044-9.
46. Perrier A, Desmarais S, Miron MJ, et al. Non-invasive diagnosis of venous thromboembolism in outpatients. Lancet 1999; 353:190-5.

47. Kucher N, Luder CM, Dornhofer T, Windecker S, Meier B, Hess OM. Novel management strategy for patients with suspected pulmonary embolism. Eur Heart J 2003; 24:366-76.
48. Kasper W, Konstantinides S, Geibel A, Tiede N, Krause T, Just H. Prognostic significance of right ventricular afterload stress detected by echocardiography in patients with clinically suspected pulmonary embolism. Heart 1997; 77:346-9.
49. Ribeiro A, Lindmarker P, Juhlin-Dannfelt A, Johnsson H, Jorfeldt L. Echocardiography Doppler in pulmonary embolism: right ventricular dysfunction as a predictor of mortality rate. Am Heart J 1997; 134:479-87.
50. Grifoni S, Olivotto I, Cecchini P, et al. Short-term clinical outcome of patients with acute pulmonary embolism, normal blood pressure, and echocardiographic right ventricular dysfunction. Circulation 2000; 101:2817-22.
51. Guidelines on diagnosis and management of acute pulmonary embolism. Task Force on Pulmonary Embolism, European Society of Cardiology. Eur Heart J 2000; 21:1301-36.
52. Douketis JD, Crowther MA, Stanton EB, Ginsberg JS. Elevated cardiac troponin levels in patients with submassive pulmonary embolism. Arch Intern Med 2002; 162:79-81.
53. Giannitsis E, Muller-Bardorff M, Kurowski V, et al. Independent prognostic value of cardiac troponin T in patients with confirmed pulmonary embolism. Circulation 2000; 102:211-17.
54. Konstantinides S, Geibel A, Olschewski M, et al. Importance of cardiac troponins I and T in risk stratification of patients with acute pulmonary embolism. Circulation 2002; 106:1263-8.
55. Pruszczyk P, Bochowicz A, Torbicki A, et al. Cardiac troponin T monitoring identifies high-risk group of normotensive patients with acute pulmonary embolism. Chest 2003; 123:1947-52.
56. Kucher N, Wallmann D, Carone A, Windecker S, Meier B, Martin HO. Incremental prognostic value of troponin I and echocardiography in patients with acute pulmonary embolism. Eur Heart J 2003; 24:1651-6.
57. Kucher N, Printzen G, Doernhoefer T, Windecker S, Meier B, Hess OM. Low pro-brain natriuretic peptide levels predict benign clinical outcome in acute pulmonary embolism. Circulation 2003; 107:1576-8.
58. Pruszczyk P, Kostrubiec M, Bochowicz A, et al. N-terminal probrain natriuretic peptide in patients with acute pulmonary embolism. Eur Respir J 2003; 22:649-53.

9
Emergency intraoperative echocardiography

Patrick J Nash and Brian P Griffin

Key points

- Emergent intraoperative echocardiography (IOE) may be requested for evaluation of an emergency case where there was not sufficient time for adequate preoperative assessment or when an emergent situation arises during an elective surgical procedure.
- Emergent IOE should be performed by a goal-orientated individual approach.
- Intraoperative hemodynamic changes may have a profound effect on valvular lesions, especially the severity of mitral regurgitation. If necessary, the hemodynamic situation should be manipulated in order to determine the true or potential severity of a valvular lesion.

Echocardiography has been used extensively in the operating room since the early 1980s, for both perioperative diagnosis and monitoring. A number of features make it the preferred cardiac imaging modality within the operating room environment. It is portable, fast, and compact; does not involve radiation; and provides both hemodynamic and anatomical data about the heart and vascular system. Intraoperative echocardiography (IOE) is used primarily within the cardiothoracic operating room but can also be a valuable tool when unexpected situations arise during noncardiac surgery, most commonly during major vascular surgery. In emergent operative cases, IOE provides vital diagnostic information for the surgeon to help guide surgical intervention. Additionally, it provides rapid anatomical and physiologic information in the event of a sudden change in hemodynamic status either before or after surgical intervention.

IOE imaging approaches

There are three imaging approaches to emergent IOE.

The transesophageal approach

Transesophageal echocardiography (TEE) is the most widely used technique for IOE. The probe is typically inserted after induction of anesthesia and tracheal intubation, before the ‘ether screen’ over the head of the patient is put in position, remaining in place for the duration of the operation. TEE imaging can be performed in tandem with surgery without interfering with the surgical field and initial surgical preparation. Pre-pump intraoperative TEE findings correlate well with surgical observations, significant

discrepancies being reported in only 2.5% of cases in one large series.[1] It is important to be aware of the following potential pitfalls when performing intraoperative TEE:

1. It can be difficult to pass the TEE probe if the operation is in progress and the ‘ether screen’ is in place.
2. Inadequate imaging is relatively uncommon but may result from abnormal anatomy where a hiatus hernia prevents adequate apposition between the probe and the anterior esophageal wall, or when prosthetic structures cause shadowing of most, if not all, critical cardiac structures.
3. Electrical interference by electrocautery or electric saws makes interpretation of all Doppler signals impossible and results in some minor distortion of two-dimensional (2-D) images. Imaging can be resumed once the diathermy or saw is discontinued.
4. No imaging is possible while the heart is at a standstill during cardiopulmonary bypass (CPB).

The epicardial approach

Epicardial IOE refers to imaging using a high-frequency (3.0–7.0 MHz) transthoracic transducer directly on the heart when the chest is open before and/or after CPB. The probe is placed in a sterile sleeve, and an acoustic interface is made with acoustic gel and a wet epicardial surface. An acoustic stand-off, such as a sterile saline bag, is necessary when imaging structures in the first centimeter below the probe, such as the anterior wall of the ascending aorta. Epicardial imaging is often superior to TEE for evaluating the ascending aorta for atheroma or focal dissection, and for assessing the interventricular septum and determining gradients across the left ventricular outflow tract. In most circumstances, epicardial imaging is performed only if TEE is not feasible (as in history of esophageal injury/bleeding/varices) or when TEE imaging is suboptimal. Standard windows for acquisition of epicardial imaging have been described.[2] The following are disadvantages of epicardial imaging.

1. It interferes with cardiac surgery.
2. There is only limited time available for imaging.
3. Imaging of structures close to the transducer may be difficult unless a good acoustic stand-off is achieved.
4. Imaging may alter hemodynamic parameters if too much force is applied when the probe is placed on the heart, causing interference with right ventricular filling.

However, in emergent situations, the benefit of obtaining vital anatomical and hemodynamic information usually offsets such relative disadvantages.

The transthoracic imaging approach

Transthoracic imaging in the operating room has a limited role. It can be used for rapid preoperative assessment either before induction of anesthesia or after chest closure when the TEE probe has been removed, typically when rapid assessment is requested to evaluate an acute hemodynamic perturbation. However, imaging is often difficult due to

inability to position the patient optimally or because of shielding of acoustic windows by chest drains and dressings.

Requirements for IOE

Equipment

Ideally, a machine that is devoted solely to IOE should be available at all times in the operating room suite. This ultrasound machine should be capable of interfacing with multiplane TEE transducers, as well as transthoracic transducers, and be capable of performing and recording M-mode, 2-D, and all Doppler modalities. Various TTE probes (3.5–7.5 MHz) should also be available. Ideally, the ultrasound machine should have the capacity for digital storage and retrieval, facilitating rapid image review and comparison between preoperative and postoperative images. Sterile sleeves and acoustic gel should be available for epicardial imaging.

Personnel

IOE is a highly demanding technical area requiring skilled personnel for its optimal use. By definition, emergent IOE is used to make critical decisions in the operating room; thus, an especially proficient and experienced operator is necessary. Persons performing emergent IOE not only require experience in TEE imaging but should also be familiar with transthoracic and epicardial imaging and with the potential surgical approaches. Skilled operators are typically cardiologists or cardiac anesthesiologists.

IOE examination

Emergent IOE should be performed by a goal-orientated approach. Firstly, the question necessitating emergent IOE must be addressed by the modality best suited to answer the query. For example, if there is concern about an intra-operative aortic dissection, epicardial imaging may be best, especially if there is difficulty in optimally visualizing the ascending aorta by TEE. The initial portion of the study should focus on thorough imaging of the aorta in multiple planes, and then a complete, yet expedient echocardiographic examination should follow. Investigation of all important structures is vital to ensure that no critical information is omitted. Furthermore, we advocate the routine use of contrast (agitated saline mixed with blood) in the right heart to ascertain the presence or absence of intracardiac shunting (most commonly, a patent foramen ovale [PFO]) for all. Once the preoperative study is completed, a record of the examination, detailing significant findings, should be made. After cessation of CPB, the same examination technique as used preoperatively should be performed. The intraoperative examination has the following aspects, that differ from a study in the echocardiographic laboratory:

1. The echocardiogram is performed simultaneously with the operation; therefore, room lighting and/or space for the machine and echocardiographer may be sub-optimal.
2. Radiofrequency interference from other devices, especially those for diathermy, may hinder acquisition of images, especially Doppler images, for long periods of time.
3. The patient's hemodynamics can change quickly in the operating room. This can have a profound effect on valvular lesions, especially the severity of mitral regurgitation. If necessary, the hemodynamic situation should be manipulated in order to determine the true or potential severity of a valvular lesion.

It is prudent to share information only once it has been verified from as many imaging planes as possible. This principle is especially pertinent to emergent IOE, as major surgical management decisions are based on the data revealed by the IOE.

Indications for emergency IOE

The indications for emergency IOE vary by the type of operation and the stage of the operation. A request for emergent IOE may be for evaluation of an emergency case where there was not sufficient time for adequate preoperative assessment or when an emergent situation arises during an elective surgical procedure (Table 9.1). Such a request may occur at any of the following four different stages during cardiac operations (the approach and likely findings at each of these stages will be discussed below):

1. induction of anesthesia and positioning of vascular access and diagnostic catheters
2. pre-pump—from the time of induction until the patient is put on CPB
3. post-pump—at the end of the main procedure after the patient is weaned from CPB
4. before transfer to the intensive care unit (ICU)—interval from immediate weaning from CPB to leaving the operating room and returning to the ICU.

Table 9.1 Indications for emergency intraoperative echocardiography

(a) Emergency surgery with insufficient time for adequate preoperative assessment
1. Cardiac surgery Acute myocardial ischemia requiring emergent revascularization Ischemic complications (ventricular septal defect, cardiac rupture, papillary muscle rupture) Acute valvular failure (endocarditis, prosthetic malfunction) Pericardial disease (tamponade)
2. Surgery of great vessels Acute aortic dissection Aortic aneurysm rupture
3. Noncardiac surgery Hemodynamic instability New electrocardiographic changes Unexpected operative findings involving the heart or great vessels
(b) Elective cardiac surgery requiring emergent intraoperative imaging

1. At induction
   Hemodynamic collapse
   New electrocardiographic changes
   Unexplained hemodynamics
2. Pre-pump
   Unexpected surgical findings
   Calcification at the cannulation site
   Hemodynamic compromise
   Positioning of diagnostic catheters
3. Post-pump
   Difficulty in weaning
   Unexpected arrhythmia New electrocardiographic changes
   Hemodynamic compromise
   Positioning of diagnostic/therapeutic aids
4. Prior to return to ICU
   Hemodynamic compromise

Different operative procedures can give rise to specific concerns, and these will be reviewed individually. In addition to its diagnostic role, IOE is used to monitor response to therapeutic maneuvers, such as 'de-airing' of the left side of the heart, in the setting of significant amounts of intracardiac air,[3] and to monitor the response to pharmacologic interventions in the setting of severe post-pump LV dysfunction.

In a consecutive series of 66 patients requiring emergent intraoperative TEE, it was requested for cardiac procedures in 70% of cases, peripheral vascular procedures in 11%, and descending aortic surgery in 5%, with the remainder consisting of a diverse collection of noncardiac procedures.[4] The most common indication for emergent IOE in this series was hemodynamic instability, with the other primary indication in cardiac surgical patients being to assist surgical decision making/planning, while in noncardiac surgery it was to evaluate patients presenting with trauma (exclude a cardiac contusion, cardiac tamponade, or traumatic aortic dissection/rupture). Five patients were evaluated for unexplained hypoxemia, with a large alveolar-to-arterial oxygen tension gradient, to exclude the possibility of a right-to-left shunt.[4] In the evaluation of hemodynamic instability, Brandt and colleagues reported that the most commonly identified abnormalities (25% of emergent IOE) were severe left ventricular (LV) dysfunction and/or new regional wall motion abnormality (RWMA), with IOE providing new information in 86% of cases. Evaluation for a possible aortic dissection was second in frequency (21%), with no relevant findings in 20% of cases.[4] Evaluation of hemodynamic instability is listed as a category 1 indication for IOE by the Task Force Report of ASE/SCA.[5]

Emergent IOE at induction of anesthesia

Problems at induction of anesthesia are rare in experienced cardiothoracic surgical centers with an experienced cardiac anesthesiology team. Nevertheless, as the complexity of cardiac surgical procedures increases, and as the severity of comorbid conditions is now greater than before, the potential for hemodynamic compromise at this stage of the

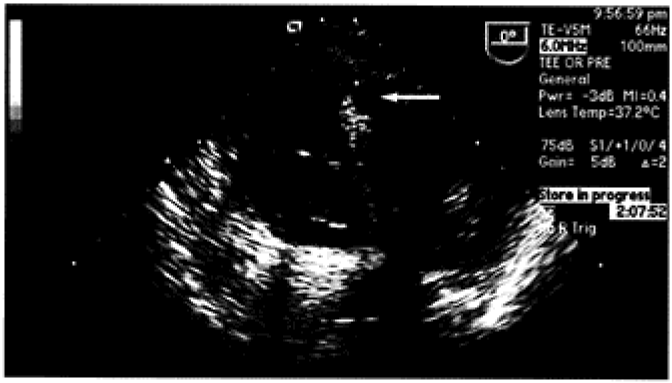
procedure remains. Several situations may arise in which the surgeon and/or anesthesiologist may request emergent IOE, typically for the rapid evaluation of hemodynamic instability. Problems typically encountered during this phase of surgery include drug reactions, myocardial ischemia, arrhythmia, and unexpected hypotension. Other less common problems encountered include a catastrophe during vascular catheter placement or the development of pericardial tamponade due to acute extension of an ascending aortic dissection. In the critical care setting, TEE has been shown to provide hemodynamic information independent of and additional to that provided by a pulmonary artery catheter.[6] Many patients undergoing cardiac surgery have tenuous hemodynamics and/or severe coronary artery disease. The hemodynamic effects of anesthetic drugs on the cardiovascular system can occasionally result in profound hemodynamic instability and/or collapse in these patients. Additionally, physiologic stresses at induction significantly increase the risk of precipitating myocardial ischemia. Continuous EGG monitoring has many limitations for the detection of early myocardial ischemia, especially in patients with known coronary artery disease and a baseline abnormal resting EGG. Data from animal and human angiographic studies show that wall motion abnormalities typically develop before EGG changes in the setting of early ischemia, and identification of left ventricular RWMA is more sensitive for the detection of compromised coronary artery blood flow than are EGG changes alone.[7,8] It is standard practice to use the 16-segment LV model with the associated coronary artery territory distributions.[9] At this phase of the operation, the TEE probe is typically not in place, and a rapidly performed transthoracic echocardiogram can often identify significant changes in global LV systolic dysfunction and/or new regional left ventricular RWMA. Frequently, however, with acute hemodynamic instability, the patient is resuscitated and ventilated and a TEE probe is passed to assess the cause of the acute decompensation.

Prepump emergency IOE

Following induction of anesthesia and mechanical ventilation, rapid deployment of the TEE probe is usually feasible. It is at this stage of surgery that emergency diagnostic imaging is most utilized. In the true emergent cardiac case, such as acute ischemia requiring surgical revascularization; acute ischemic complications, such as papillary rupture, ventricular septal defect, or cardiac rupture; or acute valvular failure from fulminant endocarditis or prosthetic valve malfunction, IOE may represent the sole means of preoperative assessment. The study should focus initially on the acute problem, but it is vital to perform a rapid, comprehensive examination of all valves, chambers, and the aorta, as additional problems may be identified that must be addressed during surgery. Additionally, a contrast injection for the detection of any right-to-left shunting, is usually performed, to detect a potential PFO, which if present and considered significant is usually closed, as there is a small but not negligible risk of hypoxemia due to right to left shunting of blood in the immediate postoperative period.

Numerous studies have shown that IOE frequently alters surgical management in 9–19% of cases, and becomes especially critical in emergent cases without routine, comprehensive preoperative assessment.[4,10–13] In other cases requiring emergent surgery where presurgical echocardiography has been performed, IOE is employed to confirm

findings, ensure no major changes in the interval, and confirm that there are no other significant additional findings that would warrant correction during this operation.



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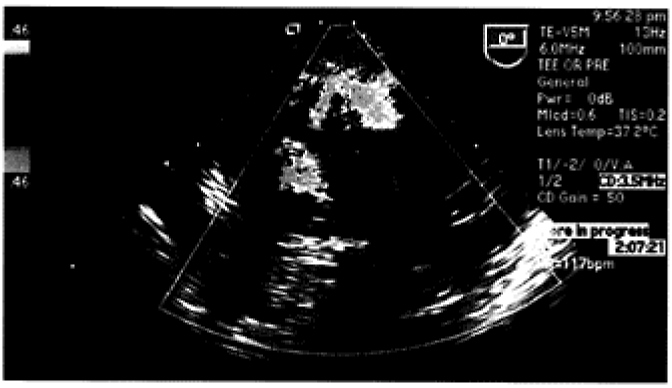

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Figure 9.1 Pre-pump intraoperative transgastric short-axis TEE image of a posterior ventricular septal rupture with left-to-right flow (arrow).

Acute myocardial ischemia

Emergent coronary revascularization is one of the most important indications for pre-pump emergent IOE. Most commonly, patients are transfers from the cardiac catheterization laboratory, after a percutaneous coronary intervention complication causing acute myocardial ischemia that is not amenable to percutaneous salvage. Occasionally, a patient with acute myocardial infarction (AMI) in whom primary percutaneous coronary intervention has failed or is not possible will present for emergent coronary artery revascularization. IOE can provide immediate information on the

distribution and extent of the affected myocardium. As with all IOE studies, a rapid, comprehensive examination should be performed, to identify any coexistent problems. This is especially pertinent in these patients, as they typically have not had an opportunity for an elective comprehensive examination, and ischemic mitral regurgitation (MR) is not an infrequent finding that may require to be addressed during the same operation. Contrast perfusion echocardiography has been used to determine the vascular bed at risk in the setting of acute ischemia. In this way, revascularization may be provided for the territory at greatest risk of ischemia initially, followed by those at lesser risk.

Mechanical complications related to myocardial ischemia

Acute mechanical complications after AMI include ventricular septal rupture, acute papillary muscle rupture, and rupture of the left ventricular free wall. Typically, the patient is hemodynamically unstable and often has had only limited and incomplete preoperative evaluation.

Ventricular septa I rupture (VSR) (Figure 9.1)

In the modern thrombolytic era, VSR has become less common (0.2% incidence, compared to 1–3% historically), occurring earlier (median of 1 day after infarction, compared to 3–5 days historically).[14] VSR occurs only where there is transmural myocardial infarction and is most common with a first infarct. It is slightly more frequent in the setting of an anterior or anterolateral infarct than an inferior wall infarct. Increasing age, female gender, history of hypertension, and lack of prior history of myocardial ischemia are associated with an increased risk of VSR.[15,16] Patients typically present with acute pulmonary edema, severe right ventricular failure, and a new murmur progressing to cardiogenic shock, and have a very poor prognosis once cardiogenic shock develops.[16] Currently, the most favored management approach is to proceed to early or emergent surgery in all patients before decompensation occurs, acknowledging that, despite such an approach, perioperative mortality remains high.[17] Typically, the diagnosis has been made prior to arrival in the operating room; however, this evaluation is often limited, and intraoperative TEE enables more accurate definition of the precise location and extent of the defect. A comprehensive color-Doppler examination of the entire interventricular septum is mandatory to localize the defect accurately, as these defects may be difficult to appreciate by 2-D imaging alone and may be multiple (‘Swiss cheese-type defect’). Typically, a VSR associated with an anterior infarct occurs in the region of the apical septum and is best visualized either in the transgastric short-axis apical view or from a midesophageal four-chamber view. Those in the setting of inferior wall infarcts more typically involve the posteroinferior basal septum and can be seen either in the basal/mid-LV transgastric short-axis view or from the four-chamber midesophageal view. Pathologically and echocardiographically, these defects are divided into two groups—simple and complex. Simple defects are those that have a direct course across the septum with one discrete color flow jet and are more frequent with anterior infarcts. Complex defects tend to be associated with a large area of myocardial necrosis, and often have multiple serpiginous defects traversing the defect (more common with large inferoposterior infarcts), and if they involve the basal septum, they may be associated

with significant mitral and/or tricuspid dysfunction.[18,19] The width of the jet has been demonstrated to correlate with the size of the defect as identified at surgery.[20] In addition to localizing the defect, TEE provides important additional information, including documenting the extent and location of LV and RV dysfunction and estimation of the RV systolic pressure. It is important to exclude other mechanical complications of the infarct, including free-wall rupture and/or papillary muscle rupture. TEE also provides important prognostic information, with those with inferoposterior defects having a higher mortality, a finding that appears to be related to the fact that these infarcts are associated with extensive RV infarction, and that these defects are more complex and challenging to repair successfully.[21,22] Those with higher right ventricular systolic pressure (RVSP) appear to have a better prognosis, probably related to the fact that they have better RV function.[21,22]

Acute ischemic MR

Hemodynamically significant acute MR can develop in the setting of an AMI for a number of reasons. Papillary muscle necrosis and rupture or partial rupture of one of the heads of the papillary muscles can result in very severe MR. Acute papillary muscle dysfunction due to extensive infarction/ischemia in the LV territory adjacent to the insertion of the papillary muscle can also result in severe MR. Also in the setting of a large infarct (most commonly anterior), LV dilation can result in a disruption of the normal geometry of the subvalvular apparatus, with apical displacement of the papillary muscles, resulting in tethering of the whole subvalvular apparatus, and causing MR. The latter two causes of MR, typically result in more chronic problems and are less likely to present emergently with acute MR in the operating room. Papillary muscle rupture typically results in very severe MR and is associated with a very poor prognosis unless there is urgent surgical intervention (Figure 9.2).[23–25] These patients develop acute pulmonary edema, hypotension, and respiratory failure. Often the murmur is relatively soft and can be missed. They are usually taken to the operating room emergently, and intraoperative TEE is performed rapidly during surgical preparation. Papillary muscle rupture most commonly occurs in the setting of a limited inferior wall myocardial infarction. The posteromedial papillary muscle is particularly vulnerable to ischemia, as it is supplied by one coronary artery, the posterior descending artery. In contrast, the anterolateral papillary muscle is supplied by both the left anterior descending and left circumflex arteries. Echocardiographically, the left ventricle is unloaded and appears small and hyperdynamic, and the area of inferior wall infarction is often limited and may easily be overlooked. Typically, the head of the papillary muscle can be seen prolapsing into the left atrium in association with torrential MR.[26] Each papillary muscle provides chordae to both leaflets, so that rupture of the posteromedial papillary muscle results in severe prolapse/flail of both leaflets (posterior aspects). The flail head of the papillary muscle is not always readily appreciated and may remain tethered or entangled within the chordae. In this situation, the presence of severe MR with a hyperdynamic LV and a short-axis view demonstrating a ‘missing’ posteromedial papillary muscle is diagnostic. In less severe cases, if only one head of the papillary muscle is torn, the resulting MR is less severe and echocardiographically requires detailed assessment of the subvalvular mitral apparatus from multiple views to confirm the underlying etiology. As the area of

infarction is often small, the long-term prognosis is relatively good if the patient survives to operation and the defect can be successfully corrected.

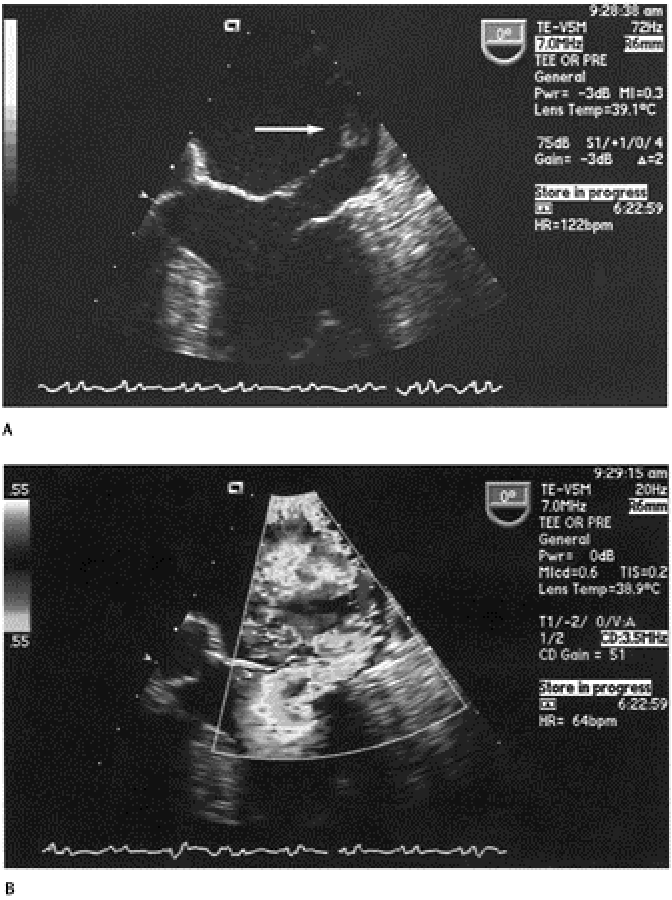


Figure 9.2 Ruptured papillary muscle with severe, posteriorly directed mitral regurgitation. Head of muscle shown by arrow.

LV free-wall rupture (Figure 9.3)

Acute rupture of the LV free wall occurs in about 3% of all transmural infarcts, causing acute tamponade, and is rapidly fatal; it is one of the primary causes of sudden death after infarction. However, in some cases, a relatively controlled rupture occurs, providing a potential window for emergent surgical intervention. This most commonly occurs in the setting of a posterolateral wall infarct, associated with left circumflex artery or left

anterior descending artery occlusion.[27,28] A nonfatal rupture typically occurs at the junction of the infarcted and normal myocardium, creating a narrow indirect channel

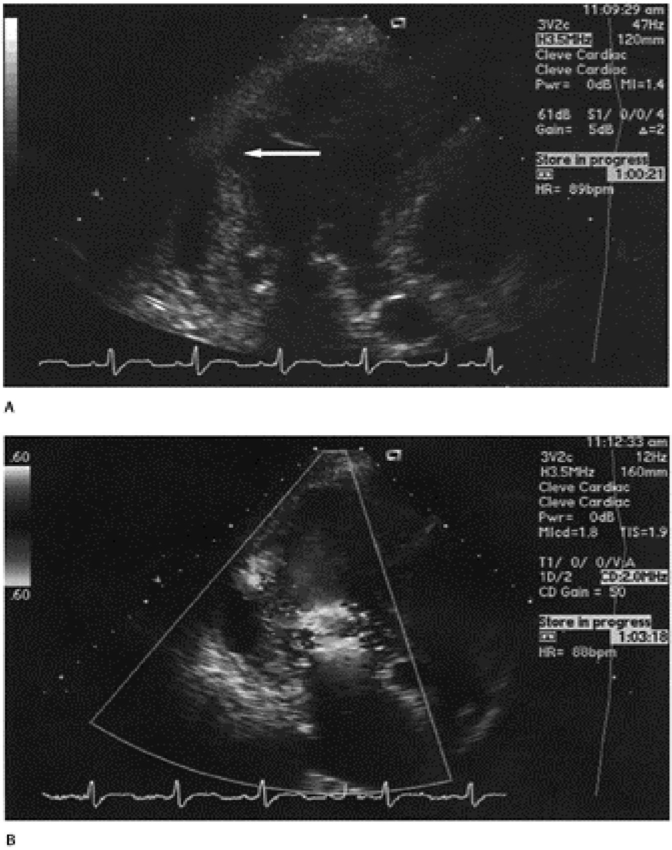


Figure 9.3 Posterior contained left ventricular free-wall rupture with pseudoaneurysm formation. Neck of aneurysm shown by arrow.

through the myocardium to the epicardium, with temporary pericardial sealing and containment, and creation of a pseudoaneurysm.[29] Typical symptoms are recurrent chest pain, hypotension, emesis, restlessness, and bradycardia. Echocardiography typically demonstrates some degree of pericardial effusion with or without the appearance of intrapericardial thrombus (appears as an echodense mass within the pericardium). This appearance in the setting of an AMI is highly specific for cardiac rupture.[29,30] Direct visualization of the communication is not always possible, although there may be the appearance of a channel by 2-D echo with some limited flow by color Doppler. Echocardiographic features suggestive of tamponade, including right atrial and/or right

ventricular diastolic collapse, respiratory variation in mitral and tricuspid inflow, and a dilated inferior vena cava, are often noted.

Acute valvular disease requiring emergent surgery

Acute MR

Patients with acute severe MR are often very ill with severe hemodynamic compromise and require urgent or emergent surgery. In the absence of acute papillary muscle rupture, acute MR may occur due to severe disruption at any level of the valve or subvalvular apparatus (Table 9.2). In the absence of ischemic heart disease, chordal rupture with resulting flail leaflet segment and severe MR can occur spontaneously in severe myxomatous valve disease or blunt chest trauma, or, very occasionally, without any apparent cause. Intraoperative echocardiographic assessment of any patient with MR involves several main goals, including assessment of MR severity, determination of the mechanism of regurgitation, evaluation of any associated features, and determination of the potential for repair. It is vital to recognize that MR is a dynamic condition, the severity of which can vary significantly depending on loading conditions. It is well recognized that intraoperative hemodynamic changes (especially changes in after-load-systolic blood pressure) can significantly reduce MR severity, and MR severity should be re-evaluated after increasing LV afterload with phenylephrine.[31,32] Valve reparability depends on mechanism and valve morphology. Repair is most likely in patients with myxomatous degeneration and is least likely in patient with valve calcification, extensive fibrosis, or leaflet destruction due to endocarditis. Feasibility of repair is greatest with posterior leaflet prolapse or flail.[33]

Acute aortic regurgitation (AR)

Causes of acute aortic regurgitation (AR) are best divided into leaflet abnormalities and aortic abnormalities (Table 9.2). Acute AR imposes a sudden acute increase in required stroke volume that the LV is required to produce, and the LV end-diastolic pressure increases acutely, causing dyspnea and pulmonary edema. Most commonly, acute AR results from an aortic dissection and will be discussed later. The role of IOE, in the setting of acute AR, is to define the anatomy of the aortic valve and ascending aorta, determine the etiology and the severity of AR, assess left ventricular function, and exclude other significant abnormalities. In the presence of relatively normal leaflets, as with an acute aortic dissection, successful repair (resuspension) of the valve is possible.

Endocarditis

Valve surgery is indicated urgently in patients with endocarditis and severe valvular regurgitation with any of the following: evidence of congestive heart failure, presence of an abscess or fistula, large vegetations (>10mm) that are either increasing or not changing in size, recurrent embolic events, and resistant infection. In the setting of acute hemodynamic instability in patients with known endocarditis, many patients may have had

Table 9.2 Causes of acute valvular regurgitation

Acute mitral regurgitation
Mitral annulus
Endocarditis with abscess
Mitral leaflet
Endocarditis—perforation or vegetation preventing complete leaflet closure
Invasive procedures—percutaneous balloon mitral valvotomy
Chordae tendineae
Myxomatous mitral valve disease
Blunt trauma or direct trauma
Endocarditis
Spontaneous
Papillary muscle
Papillary muscle rupture
Acute papillary muscle dysfunction secondary to ischemia/infarction
Trauma
Acute aortic regurgitation
Leaflet abnormalities
Traumatic rupture
Acute endocarditis
Acute prosthetic valve dysfunction
Post-aortic balloon valvuloplasty
Aortic root or ascending aorta abnormalities
Acute aortic dissection
Perivalvular leak or dehiscence of prosthetic valves

only limited opportunity to have an updated echocardiographic study to define the anatomy and current extent of infection before arriving in the operating room. A comprehensive rapid IOE examination can define the extent of infection, location and size of any abscess, involvement of or spread to other chambers, and involvement of other valves, information that is required by the surgeon to guide the operative procedure. Infection can spread from one valve to another, most commonly in the setting of a regurgitant jet of AR hitting the anterior mitral valve leaflet and setting up a satellite focus of infection, or spread can occur due to extension of infection through the fibrous skeleton of the heart. Extensive infection may lead to abscess and fistula formation. Echocardiographically, an abscess typically appears as an echolucent cavity in the perivalvular region, with limited intracavitary flow shown by color Doppler. The wall of an abscess can break down, resulting in communication with contiguous chambers, with fistula formation from either the left ventricular outflow tract (LVOT) to the RV or right atrium (Gerbode defect), or from the aorta to the left atrium or RV. Perivalvular infection

of a mitral valve can cause LV to coronary sinus fistula or spread of infection to the aortic valve. It is important to recognize these pyogenic complications of endocarditis intraoperatively, so that the surgeon may perform extensive triage and debridement. The decision to use a homograft at the aortic position is often influenced by the finding of abscess formation at the aortic valve.

Acute prosthetic valve failure

Patients with prosthetic valves may present emergently with either primary or secondary valve failure. Primary mechanical valve failure is rare, although strut failure resulting in disk embolization may occur, especially with single tilting disk valves. This is usually immediately catastrophic and rarely does a patient survive to surgery.[34] In the unusual case where the patient gets to the operating room, absence of the valve occluder and overwhelming regurgitation at the prosthetic valve is encountered. Bioprosthetic valve degeneration may lead to a sudden acute flail cusp with severe valvular regurgitation, requiring urgent surgery.[35] This is readily recognized with emergency TEE at the time of surgery. It is important in this and other acute valvular problems to try to exclude infection as a contributing cause of valve failure. Acute thrombosis on a mechanical valve can result in acute valvular dysfunction, typically valve obstruction. Emergent surgery carries significant perioperative mortality but is usually recommended in younger patients with few comorbidities and aortic or mitral valve thrombosis because of the high embolic risk attendant on thrombolysis in these patients. These patients may present emergently to surgery with minimal investigation because of extreme hemodynamic failure. TEE is sensitive and accurate in the diagnosis of prosthetic valve thrombosis,[36] typically demonstrating a valvular mass with associated obstruction of the valve occluder, Bileaflet valves such as the St Jude can present with unilateral disk obstruction. Typically, the gradients across the valve are high. Prosthetic valves are at high risk of endocarditis, with an annual rate of about 0.5% per year, with similar incidences reported for mechanical and bioprosthetic valves.[37,38] Compared to native valves, prosthetic valve endocarditis is more likely to be complicated by abscess formation and extensive perivalvular destruction, leading to the risk of valve dehiscence. Endocarditis on a bioprosthetic valve, like a native valve, can result in severe leaflet destruction and regurgitation. Perivalvular regurgitation in the setting of prosthetic valve endocarditis carries a very grave prognosis, and emergent surgery is indicated. In addition to assessing the infected valve, a comprehensive 2-D and color Doppler examination is warranted to exclude other valve involvement and document LV function.

Pericardial disease

Patients with cardiac tamponade with hemodynamic instability can present to the operating room if percutaneous drainage is not feasible or successful. In the operating room, echocardiography is used to confirm cardiac tamponade and define the location and extent of fluid, especially in patients after cardiac surgery, where loculated, localized effusions, even if small, can result in significant hemodynamic compromise. Demonstration of respiratory variation of mitral and tricuspid pulsed-wave Doppler may be difficult in the operating room setting. RV diastolic collapse (either by 2-D or M-mode

is the most specific echocardiographic sign of cardiac tamponade. Right atrial collapse is less specific. In the setting of elevated right-sided pressures in patients with pulmonary hypertension, RV collapse will not occur until higher intrapericardial pressures. In patients after cardiac surgery, loculated effusions or rapid collections of relatively small collections of blood can result in acute hemodynamic compromise. In these patients, it is important to look for atypical echocardiographic features such as left atrial collapse and/or localized echodense masses suggestive of pericardial thrombus. Echocardiography can be used for percutaneous pericardiocentesis, although, typically, in the operating room, it is as a prelude to or during open pericardial drainage.

Aortic dissection (Figure 9.4)

Acute aortic dissection is one of the more common reasons for requesting an emergent IOE. Any dissection involving the ascending aorta requires emergent surgery, as these patients have an approximately 1% per hour mortality within the first 48 h. There are a number of classifications to describe the locations of the dissection flap, but it is preferable to describe the anatomical location of the flap, and most importantly, whether it arises in the ascending aorta (Stanford type A or DeBakey type I or II—65% of acute dissections) when emergent surgery is indicated, or whether it arises in the descending aorta (Stanford type B, DeBakey type III), when medical therapy is the usual recommended strategy. Depending on the clinical status of the patient, the diagnosis of acute ascending dissection is typically made preoperatively by TEE, magnetic resonance imaging (MRI), or computed tomography (CT). However, those with suspected acute aortic dissection and acute hypotension may arrive in the operating room with minimal preoperative evaluation. Acute hypotension in the setting of a dissection is most likely due to aortic wall rupture or cardiac tamponade. Intraoperative TEE and, if necessary, epicardial imaging permit complete imaging of the

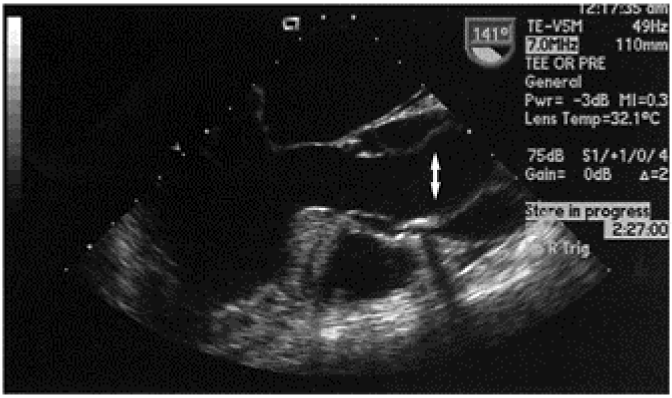


Figure 9.4 Intraoperative TEE midesophageal long-axis view of an ascending aortic dissection. Dissection flap shown by arrow.

ascending aorta, aortic valve, and LV. The TEE blind spots in the ascending aorta and transverse arch can be imaged with the epicardial probe. In addition to defining the location of the origin of the intimal flap in the ascending aorta (typically arises above the right or noncoronary cusp), and the extent of spread of the dissection in the aortic arch, great vessels, and descending aorta, IOE can comprehensively probe for important complications. Acute AR can occur due to either proximal extension of the dissection flap, resulting in disruption of leaflet suspension, or to annular dilation in the setting of patients with aortic dilation (Marfan's syndrome or annuloaortic ectasia).

From an imaging perspective, a detailed examination of the aortic valve is necessary to confirm or exclude its involvement, and predict suitability for valve repair. A prolapsing, 'normal' appearing leaflet due to involvement by the dissection of the cusp base can usually be repaired (resuspended) with a high likelihood of success.[39] In contrast, patients with aortic root dilation are typically best treated with valve replacement. Both coronary ostia should be searched for, as involvement of one or both coronary arteries by acute dissection has been estimated to occur in 10–20% of cases.[40] However, adequate visualization of the coronary ostia is not possible in all cases, with reported success rates of 88% for the left main stem and 50% for the right coronary artery.[41] If there is involvement of one of the coronary arteries (more commonly, the right coronary artery), this can result in acute ischemia in the relevant territory. Therefore, comprehensive assessment of LV function is important in these patients. In addition, a rapid assessment of all other valves and chambers is necessary, as patients may have either coexistent or related disorder (such as mitral valve prolapse in Marfan's syndrome).

Calcification at the cannulation site

Surgeons may encounter unexpected calcification of the ascending aorta. This may make placement of the bypass cannula difficult and increase the risk of embolism. Furthermore, cross-clamping of a calcified aorta may lead to catastrophic embolization of calcific material. Intraoperative aortic palpation and TEE are inaccurate methods for detecting atheroma in the ascending aorta.[42] Epicardial imaging is often necessary to provide a detailed and accurate assessment of the relevant portion of the aorta. Based on the echocardiographic appearance, the approach to cannulae insertion and/or cross-clamping may be modified, and in severe calcification, an alternate site of cannulation may be necessary, such as the subclavian artery, and the ascending aorta may need to be replaced.

Hemodynamic compromise

Acute hemodynamic compromise in the pre-pump period has precipitants similar to those at induction of anesthesia. Acute myocardial ischemia, with new LV RWMA is the most common cause, in both emergent and nonemergent cases. IOE can provide rapid, crude qualitative assessment of intravascular volume by visualization of a small, hypercontractile, 'empty' LV cavity suggestive of volume depletion. The progression of an aortic dissection causing acute aortic regurgitation or rupture can precipitate acute hemodynamic collapse. Similarly, progression of the emergent lesion requiring

intervention (enlarging VSR, progression from a partial to a complete papillary muscle rupture, valvular dehiscence, etc.) can precipitate acute

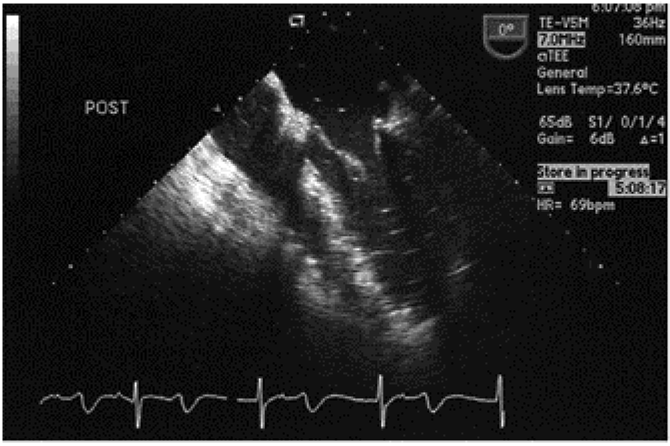


Figure 9.5 Air (bubbles) within the left ventricular cavity post-pump.

emergencies. When opening the chest, in patients with prior coronary artery bypass grafting, there is a risk of damage to previously grafted arteries, especially internal mammary grafts that often lie very close to the sternum. This may lead to acute arterial hemorrhage and acute ischemia, culminating in rapid hemodynamic compromise. Emergency echocardiography may help determine the volume status and onset of RWMA in this situation. Patients with large ascending aortic aneurysms adherent to the sternum are also at risk of iatrogenic rupture while opening the sternum. In this setting, the TEE probe is in position and the chest is typically open. IOE can rapidly make a diagnosis and/or confirm significant new changes, and the surgeons can proceed to rapid CPB to stabilize the situation.

Post-pump emergency IOE

Post-pump IOE is typically performed after the aortic cross-clamp has been removed and CPB has been weaned, but before removal of the arterial and venous CPB cannulae and prior to administration of protamine, so that if a major problem is found, CPB can be rapidly reinstituted. Routinely, post-pump IOE has the following four main aims:

1. Confirm the success of the surgical procedure (such as competency of valve repair).
2. Evaluate for intracardiac air.
3. Document satisfactory LV and RV function.
4. Evaluate for any potential complications.

Emergently, IOE can be requested for unexplained difficulty in weaning off CPB, or sudden acute hemodynamic deterioration (either unexplained hypotension or refractory malignant arrhythmia), after a transient period of hemodynamic stability after coming off pump.

Hemodynamic instability post-pump

After any cardiac operation, a variety of factors can result in hemodynamic instability and difficulty in weaning off CPB, and echocardiography is useful as both a diagnostic and a monitoring tool.[43,44] Myocardial ischemia is the most common culprit, and RWMA can usually be appreciated more easily than EGG S-T segment changes.[45] Early after cessation of CPB, there may be global LV systolic dysfunction due to residual effects of hyperkalemic cardioplegia or to widespread myocardial ischemia. A new RWMA post-pump is usually due to embolization of intracardiac air (Figure 9.5), but can be a residual effect of the cardioplegia. Air can embolize down either coronary artery, but due to its anterior position in the supine patient, the right coronary artery is most commonly affected.[46] IOE is very sensitive for the detection of intracardiac air, which is most commonly located at the LV apex, with a characteristic 'firefly' appearance, or is seen entering the left atrium from the pulmonary veins.[3,47] If very severe, the entire LV cavity may be filled with a 'snowstorm'-like effect. Typically, this improves with time but may require maintaining CPB support longer until the air has cleared and LV function is improv-ing. The surgeon will typically try to vent the air with needle puncture of the LV chamber and vent with the aortic CPB cannula until the air has dissipated.

Isolated left anterior descending and/or left circumflex territory ischemia due to air is much more unusual and gives rise to the concern that there is an intrinsic problem with the vessel or bypass graft (if grafted), external compression of the vessel, compromise of the left coronary artery ostium (especially after aortic valve or ascending aortic surgery), or, rarely, embolization of particulate material from within the heart (more likely in the setting of removal of a calcified mitral or aortic valve). The persistence of an inferior RWMA after all the air has been removed and the inferior wall has had time to recover from any air embolization should gives rise to these same concerns. For grafting immediately after coronary artery bypass, the implicated coronary artery and graft may require reinspection and possible revision of the graft if the RWMA persists. Postpump RWMA has been shown to be a significant predictor of outcome, with patients who had evidence of ischemia by IOE having adverse events in one-third of cases, compared with none in those patients with no evidence of ischemia.[48] If further revascularization is attempted by the surgeon, improvement in the RWMA may be evident by post-pump IOE if a technical difficulty in the revascularized blood vessel was responsible for the event.

In unexplained hemodynamic instability postoperatively, the ascending aorta should be screened for evidence of acute dissection, which may rarely complicate removal of the aortic CPB cannula.[49] Attention also needs to be paid to the RV, as post-pump RV dysfunction may be due to residual cardioplegia, ischemia related to inadequate cardioplegia during the CPB, air embolization down the right coronary artery, or any other cause of right coronary artery obstruction or interruption. IOE can also be used to assess intracardiac volume, and an empty hypercontractile LV in a hypotensive patient should suggest volume replacement as the initial resuscitative measure. Other potential

causes of acute hemodynamic instability are outlined in Table 9.3. A rapid assessment of all valves to document any new significant regurgitation is necessary. In the setting of unexplained hypoxemia, significant shunting can occur across a PFO, especially if right heart pressures are elevated, and this should always be looked for. The specific complications that can occur after certain cardiac procedures are outlined in Table 9.3.

Table 9.3 Potential post-pump findings in a patient with hemodynamic instability

Any cardiac surgical procedure
New regional wall motion abnormality
Major change in global LV function
Intracardiac air
Acute valvular regurgitation
Acute aortic dissection
PFO with significant right-to-left shunt
Cardiac tamponade (after closure of the chest)
Coronary artery bypass grafting (CABG)
Dissection at graft insertion
Mitral valve repair
Acute repair failure/annuloplasty ring dehiscence
LVOT obstruction due to SAM
Acute LCx occlusion due to suture
Significant residual atrial septal defect
Mitral stenosis
Valve replacement
Mitral
Aortic
Acute valve dehiscence
Mechanical strut occlusion/failure to open
Intracardiac fistula
LV pseudoaneurysm/rupture
Left atrial dissection
Strut obstruction of LVOT
Coronary ostial obstruction
Surgical myectomy
Ventricular septal defect
Residual LVOT obstruction
Residual mitral regurgitation

LV, left ventricular; PFO, patent foramen ovale; LVOT, left ventricular outflow tract; SAM, systolic anterior motion; LCx, left circumflex.

Hemodynamic instability after mitral valve repair

A decrease in LV systolic function after surgical correction of MR is not unusual, especially in those with severe MR and pre-pump subnormal LV function. This is due to unmasking of pre-pump contractile dysfunction that has been concealed by the reduced afterload associated with severe MR. Significant LVOT obstruction due to systolic anterior movement (SAM) of the mitral valve is an important complication of mitral valve repair, being reported in 3–9% of repairs.[50–53] High gradients across the LVOT can occur and result in severe hypotension, secondary MR, and inability to wean the patient from CPB. It is a dynamic condition, and its severity can be exacerbated by inotropes and/or intracardiac volume depletion. It appears to be due to anterior displacement of the mitral coaptation line, and is associated with myxomatous valve disease (especially large leaflets and those with bileaflet prolapse), the presence of a small hyperdynamic LV, and the use of a stiff annuloplasty ring.[53,54] It is easily appreciated echocardiographically, especially when severe. The gradient may be

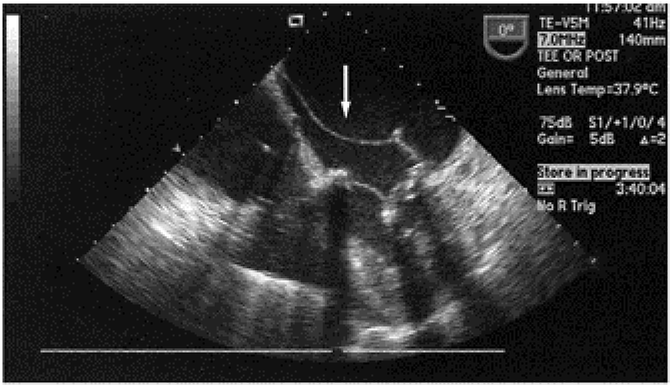


Figure 9.6 Left atrial dissection after mitral valve replacement with Carpentier-Edwards bioprosthesis. Flap of dissected atrium is shown by arrow.

difficult to measure accurately by TEE, with the best alignment usually being from a deep transgastric view. Occasionally, epicardial imaging is necessary to measure the gradient. Initial treatment involves withdrawal of inotropes and repletion of intracardiac volume, with the use of beta-blockers if feasible. If the condition persists despite these measures, a sliding annuloplasty (reduce the height of the posterior leaflet) or valve replacement may be necessary. Occasionally, suture dehiscence after repair occurs and results in severe MR. If it occurs at the site of leaflet resection in the posterior leaflet, it can simulate a posterior leaflet perforation, a complication that is easily corrected. Very occasionally, the annuloplasty ring can dehisce, and this is shown echocardiographically

by increased mobility of the ring and visualization of MR originating outside the ring. The left circumflex artery runs in the atrioventricular groove near the lateral aspect of the mitral annulus, and there is a very small risk of ensnaring it when suturing the annuloplasty ring into position. Echocardiographically, this will appear as a new and unexplained posterolateral wall motion abnormality. Finally, in limited incision mitral valve surgery, the mitral valve is typically approached through the interatrial septum, and inadequate closure may infrequently result in significant shunting at this level.

Hemodynamic instability after valve replacement

Historically, it was not uncommon to have significant ment, before the importance of preserving the subvalvular residual LV systolic dysfunction after mitral valve replaceapparatus was appreciated. Hemodynamic instability due to valve dysfunction is unusual. This can be due to primary valve dysfunction, when one or both of the disks in a mechanical valve or the leaflets in a bioprosthetic valve fail to open. This usually resolves spontaneously with increasing filling pressures. Occasionally, if suture material or residual valve apparatus is interfering with valve function, significant residual MR may result, and a second pumprun may be required to correct this. Acute valve dehiscence is extremely rare but can complicate valve replacement in the setting of severe endocarditis, especially when there is extensive perivalvular tissue destruction. A rare complication of mitral valve replacement is LV rupture or pseudoaneurysm. This rare complication can present as acute hemodynamic collapse post-pump and is more likely in patients with extensive annular calcification or those who have had repeated mitral valve surgery. A pseudoaneurysm appears as a small saccular cavity communicating with the LV cavity along the posterior aspect of the mitral valve annulus, and it requires redo surgery.[55,56] Left atrial wall dissection is a related complication with similar predisposing factors, reflected by echo in the appearance of a cavity within the left atrium with a linear echodensity consistent with a dissection flap. This can impede LV filling, or cause left atrial rupture, and it requires reoperation (Figure 9.6).[57] Rarely, after mitral bioprosthetic insertion in a patient with a small LV, LV outflow obstruction results from the struts of the bioprosthesis and is evident echocardiographically. This is increasingly rare with the advent of lower profile mitral bioprostheses.

Hemodynamic impairment after myectomy

After myectomy, hemodynamic impairment may result from an inadequate resection of muscle with residual LV outflow obstruction and systolic anterior mitral valve motion. This usually requires further removal of muscle, or if this is impossible, mitral valve replacement with a low-profile valve. If the resection of muscle is too large, ventricular septal defect with left-to-right shunting may result. This may be difficult to detect without utilizing multiple views and may even require epicardial imaging for detection. Septal perforator transection is normal after myectomy; it consists of a low-velocity flow from the septum into the LV and should not be mistaken for ventricular septal defect. Patients with hypertrophic cardiomyopathy are at risk of LV dysfunction postoperatively, especially if the procedure is prolonged and myocardial preservation is inadequate.

Emergent IOE before leaving the operating room

Hemodynamic instability after the chest is closed and the patient is being prepared for transfer to the ICU is typically due to factors similar to those immediately post-pump. At this stage, the TEE probe typically has been removed. An initial approach may be to try to screen for potential problems with transthoracic imaging; however, this is often very difficult in patients after surgery, especially with impediments such as chest drains and bandages impeding the conventional imaging windows. If necessary, the TEE probe may need to be reinserted. Intracardiac air is unlikely to be the culprit, unless significant residual air was seen on final post-pump imaging. Chest closure can change the anatomical orientation to the heart and more importantly of bypass grafts, and there is a risk of graft kinking or compression. A more likely cause of acute hemodynamic compromise is cardiac tamponade. This may be heralded by increased drainage through the chest tubes. A localized collection at a critical point (such as behind the left atrium) or the rapid collection of a relatively small volume of fluid can cause disproportionate hemodynamic compromise.

Emergent IOE for placement of supporting devices

Emergent IOE may be requested to assist with the placement of intravascular catheters and cardiac assist devices. In the operating room, IOE can be of assistance in visualizing the location of a pulmonary artery catheter that cannot be easily passed into the correct position in the pulmonary artery, and can demonstrate coiling in the right atrium or ventricle. However, it is difficult to visualize adequately the 3-D location of the catheter by 2-D echocardiography; in addition, the RVOT is in the far field, and it and the pulmonary arteries are not always easy to visualize optimally by TEE. Intraoperative TEE is often used to confirm the location of the distal tip of the balloon of an intra-aortic balloon pump when inserted in the operating room without fluoroscopy. The correct location of the tip is at the junction of the aortic arch and descending aorta after the origin of the left subclavian artery.

Ventricular assist devices

Before implantation of a LV assist device (LVAD), IOE should focus on a number of important areas which provide vital information for the surgeon, prior to proceeding with the surgery.[58] Severe RV dysfunction gives rise to concerns that the RV will be unable to maintain sufficient forward cardiac output and filling of the left heart, and that an additional RV assist device (RVAD) will be required. The pump inflow cannula is typically placed in the LV apex with the pump outflow cannula returning blood to the ascending aorta. Evaluation of the LV apex for thrombus and the ascending aorta for atheroma is important. Additionally, the presence and severity of AR is important. Severe AR results in futile reverse flow of blood, reducing the efficacy of the pump. If this disorder is significant, corrective aortic valve surgery will be required. Finally, if a PFO is identified, it should be closed, because when the pump is implanted and left atrial pressure falls, blood can be shunted from right to left across the PFO. Immediately after

implantation of an LVAD, IOE should show the aortic valve either not opening or opening infrequently as it is effectively bypassed. Flows into the inflow and from the outflow cannula should be documented. Typically, flows should be less than 2 m/s for both. Increased flow velocity at the inflow cannula may be due to angulation and partial obstruction of the cannula, and may require repositioning if this is significantly affecting pump flow. Otherwise, the post-pump IOE examination should evaluate RV function, confirm no evidence of a PFO, and exclude evidence of aortic dissection.

Indications for emergent IOE during noncardiac surgery

Emergency echocardiography is occasionally requested during noncardiac surgery. In most instances, patients undergoing noncardiac surgery have had a diagnostic work-up to exclude significant underlying cardiac problems, especially if there is a suggestion from the present or past history that the patient's cardiovascular status may be of concern. However, unexpected situations do arise even in the best-planned and -executed procedures. The noncardiac procedures where echocardiography is likely to be required usually are vascular procedures, especially those on the thoracic or abdominal aorta; thoracic procedures involving lung carcinoma; surgery to remove suspected renal carcinoma; and major gastrointestinal operations. Most commonly, IOE is requested in the setting of hemodynamic instability, which, for nonthoracic surgery, is typically due to acute myocardial ischemia precipitated by acute changes in intravascular volume and/or blood pressure in patients undergoing major vascular surgical procedures, such as repair of abdominal aortic aneurysms. The findings are as described for similar changes during cardiac procedures. During major vascular surgery, reduction in preload from reduction in venous return and increase in afterload from aortic cross-clamping may lead to severe afterload mismatch, with severe reduction in ventricular contractile function. This is especially the case with suprarenal aortic surgery. These findings may be exacerbated by acidosis from tissue underperfusion in this setting. Distinguishing this situation from severe ischemia may be difficult, but the global rather than regional effect on ventricular function is helpful in differentiation. Improvement in function usually occurs in response to removal of the aortic cross-clamp. Occasionally during abdominal or thoracic surgery, extension of a disease process from these cavities into the mediastinum and cardiac chambers or great vessels requires emergency diagnostic TEE. Conditions in which this may be required include lung tumors invading the heart directly or via the pulmonary veins, or renal tumors propagating along the inferior vena cava.

Influence of IOE on patient management

IOE has been demonstrated to have a significant impact on patient management. In a large series (over 3000 predominantly elective surgical patients), Click and colleagues reported that new information was found on pre-pump intraoperative TEE that directly affected surgery in 14% of patients, primarily the identification of a PFO that resulted in closure in the majority of patients.[12] On post-pump studies, new information was found after surgery in 6% of patients, which resulted in a change in surgery or hemodynamic

management in 4%.[12] Brandt et al reported on 66 patients who underwent emergent IOE, reporting that TEE showed new information in most (80%), and in 15 (23%) resulted in an alteration to the planned surgical procedure.[4]

Conclusions and recommendations

Emergent IOE is a powerful diagnostic tool that has wide applicability to various operating room environments. The challenge to the operator is to utilize quickly the best available modality that has the highest likelihood of answering the anatomical or hemodynamic questions that arise during surgery. As new technological developments continue to change and improve both the surgical and echocardiographic specialties, the operator will need to strive to ensure similar adaptive changes; this will require practicality, frugality, and creativity when employing emergent IOE. Emergency IOE will probably have an increasingly important role in the operating room in the future. As the complexity of cardiac and noncardiac surgery increases, and operative patients continue to become older and sicker, there will be a greater role for the rapid, accurate anatomical and hemodynamic data that echocardiography provides.

References

1. Chaliki HP, Click RL, Abel MD. Comparison of intraoperative transesophageal echocardiographic examinations with the operative findings: prospective review of 1918 cases. J Am Soc Echocardiogr 1999; 12:237–40.
2. Stewart WJ, Currie PJ, Agler DA, Cosgrove DM. Intraoperative epicardial echocardiography: technique, imaging planes and use in valve repair for mitral regurgitation. Dynamic Cardiovasc Imaging 1987:166.
3. Duff HJ, Buda AJ, Kramer R, Strauss HD, David TE, Berman ND. Detection of entrapped intracardiac air with intraoperative echocardiography. Am J Cardiol 1980; 46:255–60.
4. Brandt RR, Oh JK, Abel MD, Click RL, Orszulak TA, Seward JB. Role of emergency intraoperative transesophageal echocardiography. J Am Soc Echocardiogr 1998; 11:972–7.
5. Cahalan MK, Stewart W, Pearlman A, et al. American Society of Echocardiography and Society of Cardiovascular Anesthesiologists task force guidelines for training in perioperative echocardiography. J Am Soc Echocardiogr 2002; 15:647–52.
6. Poelaert JI, Trouerbach J, De Buyzere M, Everaert J, Colardyn FA. Evaluation of transesophageal echocardiography as a diagnostic and therapeutic aid in a critical care setting. Chest 1995; 107:774–9.
7. Wohlgelernter D, Jaffe CC, Cabin HS, Yeatman LA Jr, Cleman M. Silent ischemia during coronary occlusion produced by balloon inflation: relation to regional myocardial dysfunction. J Am Coll Cardiol 1987; 10:491–8.
8. Battler A, Froelicher VF, Gallagher KP, Kemper WS, Ross J. Dissociation between regional myocardial dysfunction and ECG changes during ischemia in the conscious dog. Circulation 1980; 62:735–44.
9. Schiller NB, Shah PM, Crawford M, et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. J Am Soc Echocardiogr 1989; 2:358–67.

10. Sheikh KH, Bengtson JR, Rankin JS, de Bruiin NP, Kisslo J. Intra-operative transesophageal Doppler color flow imaging used to guide patient selection and operative treatment of ischemic mitral regurgitation. Circulation 1991; 84:594–604.
11. Michel-Cherqui M, Ceddaha A, Liu N, et al. Assessment of systematic use of intraoperative transesophageal echocardiography during cardiac surgery in adults: a prospective study of 203 patients. J Cardiothorac Vasc Anesth 2000; 14:45–50.
12. Click RL, Abel MD, Schaff HV. Intraoperative transesophageal echocardiography: 5-year prospective review of impact on surgical management. Mayo Clin Proc 2000; 75:241–7.
13. Mishra M, Chauhan R, Sharma KK, et al. Real-time intraoperative transesophageal echocardiography—how useful? Experience of 5,016 cases. J Cardiothorac Vasc Anesth 1998; 12:625–32.
14. Crenshaw BS, Granger CB, Birnbaum Y, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. GUSTO-I (Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries) Trial Investigators. Circulation 2000; 101:27–32.
15. Lemery R, Smith HC, Giuliani ER, Gersh BJ. Prognosis in rupture of the ventricular septum after acute myocardial infarction and role of early surgical intervention. Am J Cardiol 1992; 70:147–51.
16. Birnbaum Y, Fishbein MC, Blanche C, Siegel RJ. Ventricular septal rupture after acute myocardial infarction. N Engl J Med 2002; 347:1426–32.
17. Ryan TJ, Antman EM, Brooks NH, et al. 1999 update: ACC/AHA guidelines for the management of patients with acute myocardial infarction. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). J Am Coll Cardiol 1999; 34:890–911.
18. Edwards BS, Edwards WD, Edwards JE. Ventricular septal rupture complicating acute myocardial infarction: identification of simple and complex types in 53 autopsied hearts. Am J Cardiol 1984; 54:1201–5.
19. Obarski TP, Rogers PJ, Debaets DL, Murcko LG, Jennings MR. Assessment of postinfarction ventricular septal ruptures by transesophageal Doppler echocardiography. J Am Soc Echocardiogr 1995; 8:728–34.
20. Helmcke F, Mahan EF 3rd, Nanda NC, et al. Two-dimensional echocardiography and Doppler color flow mapping in the diagnosis and prognosis of ventricular septal rupture. Circulation 1990; 81:1775–83.
21. Menon V, Webb JG, Hillis LD, et al. Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: a report from the SHOCK Trial Registry. Should we emergently revascularize occluded coronaries in cardiogenic shock? J Am Coll Cardiol 2000; 36:1110–16.
22. Moore CA, Nygaard TW, Kaiser DL, Cooper AA, Gibson RS. Postinfarction ventricular septal rupture: the importance of location of infarction and right ventricular function in determining survival. Circulation 1986; 74:45–55.
23. Replogle RL, Campbell CD. Surgery for mitral regurgitation associated with ischemic heart disease. Results and strategies. Circulation 1989; 79:I122–5.
24. Nishimura RA, Schaff HV, Gersh BJ, Holmes DR Jr, Tajik AJ. Early repair of mechanical complications after acute myocardial infarction. JAMA 1986; 256:47–50.
25. Nishimura RA, Schaff HV, Shub C, Gersh BJ, Edwards WD, Tajik AJ. Papillary muscle rupture complicating acute myocardial infarction: analysis of 17 patients. Am J Cardiol 1983; 51:373–7.
26. Brack M, Asinger RW, Sharkey SW, Herzog CA, Hodges M. Two-dimensional echocardiographic characteristics of pericardial hematoma secondary to left ventricular free wall rupture complicating acute myocardial infarction. Am J Cardiol 1991; 68:961–4.
27. Slater J, Brown RJ, Antonelli TA, et al. Cardiogenic shock due to cardiac free-wall rupture or tamponade after acute myocardial infarction: a report from the SHOCK Trial Registry. Should

we emergently revascularize occluded coronaries for cardiogenic shock? J Am Coll Cardiol 2000; 36:1117–22.
28. Oliva PB, Hammill SC, Edwards WD. Cardiac rupture, a clinically predictable complication of acute myocardial infarction: report of 70 cases with clinicopathologic correlations. J Am Coll Cardiol 1993; 22:720–6.
29. Lopez-Sendon J, Gonzalez A, Lopez de Sa E, et al. Diagnosis of subacute ventricular wall rupture after acute myocardial infarction: sensitivity and specificity of clinical, hemodynamic and echocardiographic criteria. J Am Coll Cardiol 1992; 19:1145–53.
30. Raitt MH, Kraft CD, Gardner CJ, Pearlman AS, Otto CM. Subacute ventricular free wall rupture complicating myocardial infarction. Am Heart J 1993; 126:946–55.
31. Grewal KS, Malkowski MJ, Piracha AR, et al. Effect of general anesthesia on the severity of mitral regurgitation by transesophageal echocardiography. Am J Cardiol 2000; 85:199–203.
32. Konstadt SN, Louie EK, Shore-Lesserson L, Black S, Scanlon P. The effects of loading changes on intraoperative Doppler assessment of mitral regurgitation. J Cardiothorac Vasc Anesth 1994; 8:19–23.
33. Cosgrove DM, Stewart WJ. Mitral valvuloplasty. Curr Probl Cardiol 1989; 14:359–415.
34. Grunkemeier GL, Starr A, Rahimtoola SH. Prosthetic heart valve performance: long-term follow-up. Curr Probl Cardiol 1992; 17:329–406.
35. Bansal RC, Morrison DL, Jacobson JG. Echocardiography of porcine aortic prosthesis with flail leaflets due to degeneration and calcification. Am Heart J 1984; 107:591–3.
36. Dzavik V, Cohen G, Chan KL. Role of transesophageal echocardiography in the diagnosis and management of prosthetic valve thrombosis. J Am Coll Cardiol 1991; 18:1829–33.
37. Hammermeister KE, Henderson WG, Burchfiel CM, et al. Comparison of outcome after valve replacement with a bioprosthesis versus a mechanical prosthesis: initial 5 year results of a randomized trial. J Am Coll Cardiol 1987; 10:719–32.
38. Bloomfield P, Wheatley DJ, Prescott RJ, Miller HC. Twelve-year comparison of a Bjork-Shiley mechanical heart valve with porcine bioprostheses. N Engl J Med 1991; 324:573–9.
39. Movsowitz HD, Levine RA, Hilgenberg AD, Isselbacher EM. Transesophageal echocardiographic description of the mechanisms of aortic regurgitation in acute type A aortic dissection: implications for aortic valve repair. J Am Coll Cardiol 2000; 36:884–90.
40. Crawford ES. The diagnosis and management of aortic dissection. JAMA 1990; 264:2537–41.
41. Ballal RS, Nanda NC, Gatewood R, et al. Usefulness of transesophageal echocardiography in assessment of aortic dissection. Circulation 1991; 84:1903–14.
42. Royse C, Royse A, Blake D, Grigg L. Screening the thoracic aorta for atheroma: a comparison of manual palpation, transesophageal and epiaortic ultrasonography. Ann Thorac Cardiovasc Surg 1998; 4:347–50.
43. Chan KL. Transesophageal echocardiography for assessing cause of hypotension after cardiac surgery. Am J Cardiol 1988; 62:1142–3.
44. Reichert CL, Visser CA, Koolen JJ, et al. Transesophageal echocardiography in hypotensive patients after cardiac operations. Comparison with hemodynamic parameters. J Thorac Cardiovasc Surg 1992; 104:321–6.
45. Smith JS, Cahalan MK, Benefiel DJ, et al. Intraoperative detection of myocardial ischemia in high-risk patients: electrocardiography versus two-dimensional transesophageal echocardiography. Circulation 1985; 72:1015–21.
46. Obarski TP, Loop FD, Cosgrove DM, Lytle BW, Stewart WJ. Frequency of acute myocardial infarction in valve repairs versus valve replacement for pure mitral regurgitation. Am J Cardiol 1990; 65:887–90.
47. Orihashi K, Matsuura Y, Hamanaka Y, et al. Retained intracardiac air in open heart operations examined by transesophageal echocardiography. Ann Thorac Surg 1993; 55:1467–71.
48. Leung JM, O’Kelly B, Browner WS, Tubau J, Hollenberg M, Mangano DT. Prognostic importance of postbypass regional wall-motion abnormalities in patients undergoing coronary artery bypass graft surgery. SPI Research Group. Anesthesiology 1989; 71:16–25.

49. Katz ES, Tunick PA, Colvin SB, Culliford AT, Kronzon I. Aortic dissection complicating cardiac surgery: diagnosis by intraoperative biplane transesophageal echocardiography. J Am Soc Echocardiogr 1993; 6:217–22.
50. Marwick TH, Stewart WJ, Currie PJ, Cosgrove DM. Mechanisms of failure of mitral valve repair: an echocardiographic study. Am Heart J 1991; 122:149–56.
51. Kronzon I, Cohen ML, Winer HE, Colvin SB. Left ventricular outflow obstruction: a complication of mitral valvuloplasty. J Am Coll Cardiol 1984; 4:825–8.
52. Kreindel MS, Schiavone WA, Lever HM, Cosgrove D. Systolic anterior motion of the mitral valve after Carpentier ring valvuloplasty for mitral valve prolapse. Am J Cardiol 1986; 57:408–12.
53. Lee KS, Stewart WJ, Lever HM, Underwood PL, Cosgrove DM. Mechanism of outflow tract obstruction causing failed mitral valve repair. Anterior displacement of leaflet coaptation. Circulation 1993; 88:1124–9.
54. Freeman WK, Schaff HV, Khandheria BK, et al. Intraoperative evaluation of mitral valve regurgitation and repair by transesophageal echocardiography: incidence and significance of systolic anterior motion. J Am Coll Cardiol 1992; 20:599–609.
55. Karlson KJ, Ashraf MM, Berger RL. Rupture of left ventricle following mitral valve replacement. Ann Thorac Surg 1988; 46:590–7.
56. Ballal R, Nanda NC, Sanyal R. Intraoperative transesophageal echocardiographic diagnosis of left atrial pseudoaneurysm. Am Heart J 1992; 123:217–18.
57. Lukacs L, Kassai I, Lengyel M. Dissection of the atrial wall after mitral valve replacement. Tex Heart Inst J 1996; 23:62–4.
58. Scalia GM, McCarthy PM, Savage RM, Smedira NG, Thomas JD. Clinical utility of echocardiography in the management of implantable ventricular assist devices. J Am Soc Echocardiogr 2000; 13:754–63.

10
The role of echocardiography in primary percutaneous coronary intervention for acute myocardial infarction

Leonardo Bolognese and Giampaolo Cerisano

Key points

- After primary angioplasty for acute myocardial infarction, improved tissue level perfusion is related to improved clinical outcome independently of the flow in epicardial infarct-related artery.
- Myocardial contrast echocardiography (MCE) is promising method of assessing microvascular integrity and myocardial viability after acute myocardial infarction.
- The spatial distribution of microbubbles directly injected through infarct-related coronary artery into the myocardium provides an assessment of regional capillary integrity.
- Intracoronary MCE can detect the extent of myocardium within the risk area in which the microvasculature has not been destroyed by prolonged ischemia, and which consequently may show late functional improvement.
- Intravenous MCE can potentially increase its clinical applications; however, there are still significant methodological, interpretative, and training issues to be resolved.

In the absence of an adequate collateral flow, occlusion of an epicardial coronary artery leads to myocardial ischemia and myocardial necrosis if the reduction of flow is sustained enough to produce irreversible damage.[1] Timely reperfusion, either spontaneous or obtained by thrombolysis or percutaneous coronary intervention (PCI), mediates myocardial salvage, thereby limiting the wave front and the extension of myocardial necrosis, while strongly affecting short and long-term prognosis. Thus, the restoration of full patency is the prerequisite to avoid or limit myocardial infarction, and it mediates left ventricular volume and survival after acute myocardial infarction (AMI). Correlations between the sustained patency of the infarct-related artery and improved clinical outcomes culminated in the ‘open-artery hypothesis’ which has been the cornerstone of therapeutic strategies for AMI for over a decade. However, the open-artery hypothesis may be an oversimplification, because the goal of reperfusion should be to restore not only upstream epicardial patency and flow but also downstream myocardial tissue perfusion. Microvascular dysfunction after reperfusion therapy may, in fact, profoundly affect the function of cardiac myocytes and interstitium, with important pathophysiologic and prognostic implications.[2,3] Nowadays, perhaps, ‘open vasculature hypothesis’ is a more appropriate term than ‘open-artery hypothesis’, because it has been

shown that improved tissue-level perfusion is related to improved clinical outcome independently of flow in the epicardial artery.[4] The downstream shifting of the open-artery hypothesis is expected to redefine the goals of reperfusion strategies to include not only rapid and sustained epicardial patency but also restored microvascular flow and myocardial tissue perfusion.

Therefore, in patients with AMI undergoing primary PCI, the following three questions need to be promptly answered:

1. Has the myocardium been optimally reperfused?
2. How much of the myocardium has been salvaged and how much has the potential to recover further during follow-up?

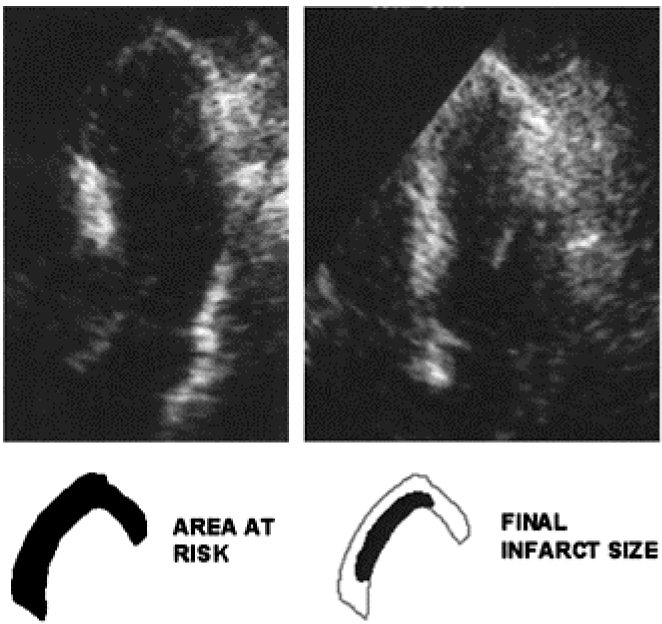


Figure 10.1 Serial evaluation of contrast enhancement in the territory of the occluded artery allows the assessment of risk area and final infarct size.

3. Is monitoring of left ventricular function able to identify meaningful predictors of clinical outcome that are independent of the perfusion status of the infarctrelated artery?

Echocardiography has the potential adequately to address all these three main issues. The major promise of the technique in the setting of AMI is to provide reliable information on regional function and perfusion and flow reserve, an achievement that would bring

echocardiography close to the Holy Grail of the ‘one-stop shopping’ technique claimed by other modalities. This chapter critically assesses whether this promise has been fulfilled in the setting of AMI treated by means of primary PCI, and, most importantly, whether echocardiography has become an important clinical tool that may help in the decision making for management of these patients in routine practice.

Assessment of microvascular dysfunction during primary PCI

Until recently, we have had limited access to diagnose microvascular obstruction in living patients and therefore to assess the effects of different therapeutic tools on microcirculation. With the availability of imaging technology, microvascular dysfunction has been documented in a far greater proportion of patients than ever conceived.

There are a number of methods for assessing tissue-level myocardial reperfusion including angiographic, echocardiographic, and nuclear techniques, as well as simple markers such as ST-segment resolution. Myocardial contrast echocardiography (MCE) is gaining interest as a promising method of assessing microvascular integrity and myocardial viability after AMI. MCE uses microbubbles that remain in the intravascular space. Because 90% of the microvasculature consists of capillaries,[5] the spatial distribution of these bubbles in the myocardium provides an assessment of regional capillary integrity. Normal microvascular perfusion is present in regions of viable myocardium, whereas regions of necrosis do not demonstrate microvascular perfusion or show poor perfusion.[6,7] Thus, after successful reperfusion, MCE can detect the extent of myocardium within the risk area in which the microvasculature has not been destroyed by prolonged ischemia and which consequently may show late functional improvement. Three different perfusion patterns in the infarct bed have been described after reperfusion: no opacification, patchy or intermediate opacifi-

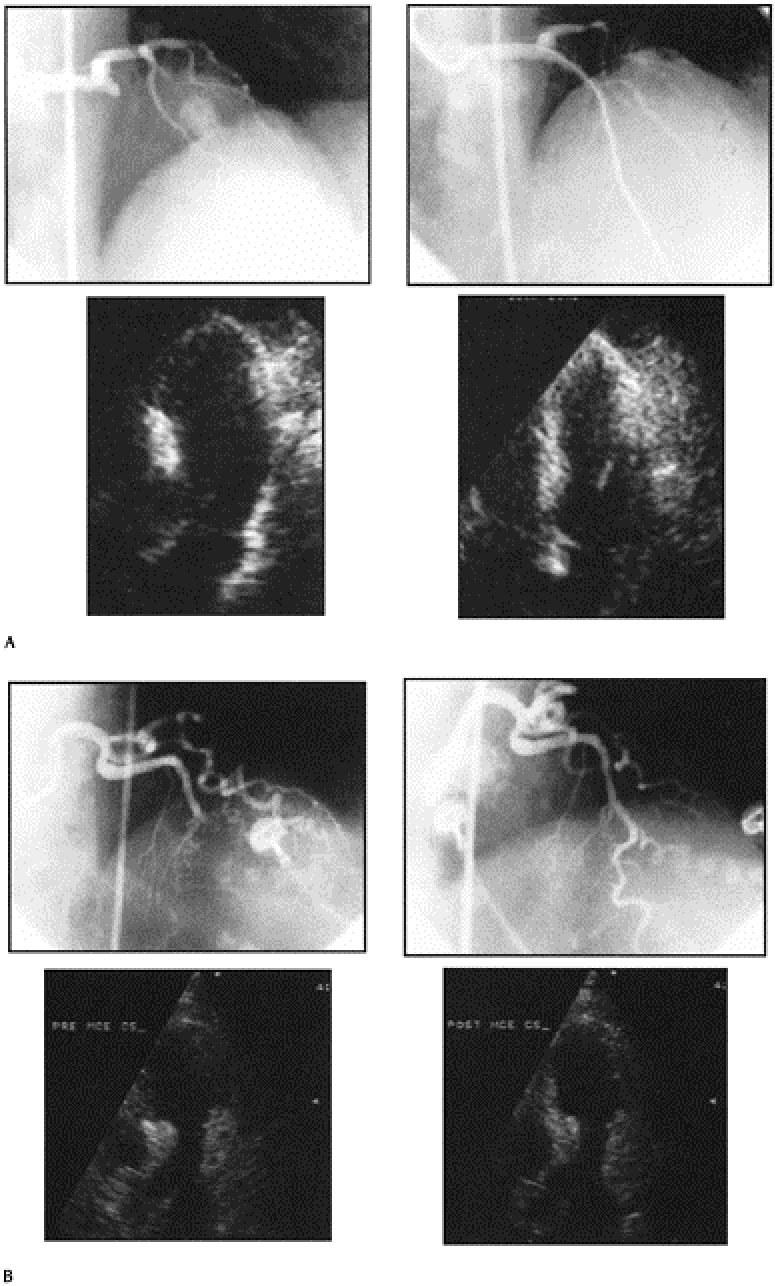


Figure 10.2 (A) Intracoronary myocardial contrast echocardiography (MCE) before (left), and after (right)

recanalization of the infarct-related left anterior descending coronary artery, showing homogeneous contrast enhancement in the risk area. (B) Myocardial contrast echocardiography before (left panels) and after (right panels) recanalization of the infarct-related left anterior descending artery. No contrast enhancement (lower right panel) is visible after successful recanalization of the vessel indicating no reflow.

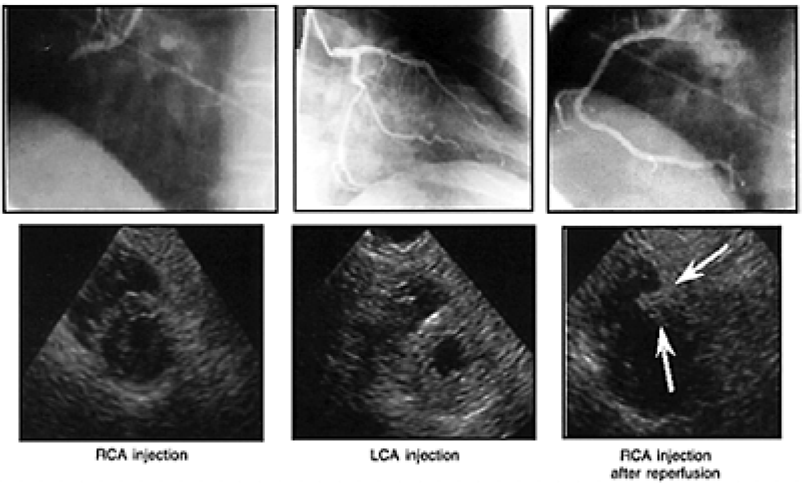


Figure 10.3 Patient with an inferior AMI due to the proximal occlusion of the right coronary artery (RCA) treated with primary PCI. The direct intracoronary injection of the contrast agent in the RCA does not produce contrast enhancement in the territory of RCA (left panel). Left coronary angiogram (LCA) does not show angiographic collateral circulation to RCA, but the MCE image shows a quite homogeneous contrast

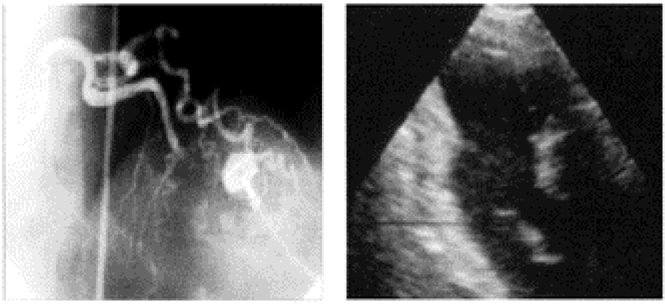
enhancement involving also the RCA territory of distribution (middle panel). Finally, the intracoronary injection of the contrast agent in the RCA after optimal recanalization of the vessel produces a significant contrast enhancement in the posteroseptal and inferior segments as well as in the free wall of the right ventricle (arrows, right panel).

cation (including also opacification noted only in the epicardium), and homogeneous opacification.[8] Dysfunctional regions with extensive myocardial opacification show near normal function at follow-up, while those with no opacification show the most dysfunction. Areas where the spatial extent of microvascular perfusion was intermediate or patchy show intermediate function at followup.[8] Thus, MCE has the potential to provide an optimal assessment of microvascular integrity and viability in patients with AMI undergoing reperfusion therapy.

Two different approaches may be used to obtain this information. The first one implies the use of intracoronary MCE during primary PCI. Taking advantage of the access to the coronary circulation offered during angioplasty, this technique relies on direct intracoronary injection of contrast agents containing microbubbles, often using simple, sonicated, radiopaque dyes. More recently, it has been possible to opacify the myocardium with intravenous injection of contrast.[9,10] This approach has the potential to increase the clinical applications of MCE, if this technique is used directly in coronary care unit.

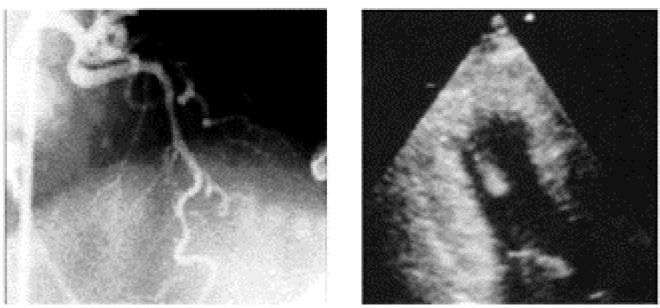
The assessment of microvascular integrity by intracoronary MCE has been investigated by several authors.[8,11–15] By this approach it is possible to define the risk area and final infarct size, to assess the success of reperfusion, and to quantify the extent of salvaged myocardium. The main messages of these studies are as follows:

1. MCE is a reliable method of identifying myocardium at risk of necrosis, as well as final infarct size (Figure 10.1).
2. It can assess tissue perfusion and integrity, unequivocally showing that reflow in the infarct-related artery does not necessarily imply tissue reperfusion (Figure 10.2A and 10.2B).
3. It can provide accurate mapping of the extension of



A

Figure 10.4A Left coronary angiography (left); MCE long-axis view in a patient with acute anterior myocardial infarction before recanalization (right). The angiogram shows occlusion of the left anterior descending coronary artery in the midportion after the first diagonal branch. MCE reveals the absence of opacification in the medium segment of the septum and apex (area at risk).



B

Figure 10.4B Repeat MCE shortly after successful PCI of left anterior descending coronary artery (left) shows homogeneous contrast enhancement of the risk area (right).

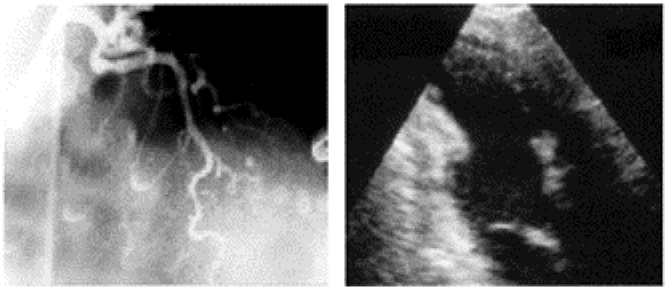


Figure 10.4C Forty-eight-hours follow-up coronary angiography (left) showing the persistence of an optimal epicardial patency. MCE (right) reveals the disappearance of the significant contrast enhancement in the risk area.

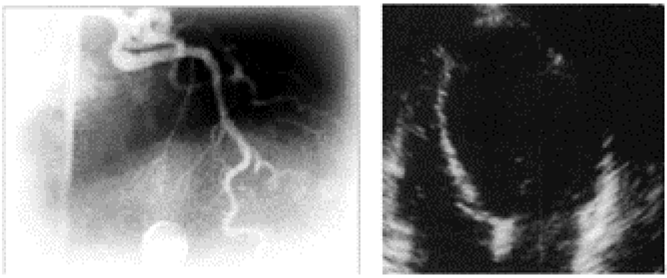


Figure 10.4D Four weeks after discharge: repeat coronary angiography demonstrates the persistence of left anterior descending coronary artery patency (left), but two-dimensional echocardiography (four-chamber view) shows a dilated, poorly contracting left ventricle (right).

functionally effective intramyocardial collateral circulation (Figure 10.3).

4. Preservation of microcirculation after reperfusion is a prerequisite for functional recovery, prevents left ventricular remodeling,[10,16–18] and favorably affects the clinical outcome.[14,19]

Although exciting, these results should be interpreted with caution. Because of hyperemia during the early hours of reperfusion,[20] MCE may underestimate infarct size and overestimate myocardial salvage.[21] This may be particularly true when the residual stenosis within the infarct-related artery is not severe enough to attenuate the hyperemic flow. The use of an exogenously administered coronary vasodilator, such as dypiridamole, to unmask the reduced microvascular reserve of the infarcted tissue has been recently proposed.[22] However, this approach may be unpractical or even unsafe during coronary angioplasty performed for the treatment of an evolving AMI. Furthermore, in all studies, MCE, in spite of very high sensitivity in predicting functional recovery after reperfusion, shows very low specificity, ranging from 15% to 60%.[14,15,23–25] Thus, contrast enhancement within the area at risk shortly or late after reperfusion, even if homogeneous, does not necessarily imply regional functional recovery in the chronic stage. Recent studies, which have compared microvascular integrity assessed by MCE with contractile reserve in the infarcted area elicited by dobutamine infusion, have shown that, whereas the absence of intramyocardial perfusion is strictly related to irreversible functional damage, microvascular integrity can be present not only in segments with contractile reserve and functional recovery but also in segments without such characteristics.[23–26] Similar results have also been achieved by comparing MCE with other imaging techniques that explore cellular integrity such as sestamibi SPECT.[27]

There are several factors, besides the effect of hyperemia on MCE performance, that may help in interpreting these findings. The infarct area may be a mixing of islands of viable and necrotic myocytes.[28] In this scenario, it is possible that perfusion, although to a lesser extent than normal regions, may be detected although functional integrity is precluded by an insufficient number of viable myocytes. This hypothesis might be suggested by the patchy pattern of contrast enhancement, which appears to be a less specific marker of myocardial viability than the homogeneous pattern.[23] Finally, a temporal dissociation between microvascular and cellular damage may exist,[29] and recent studies have suggested that late evaluation of myocardial perfusion by MCE correlates with late functional recovery more closely than myocardial perfusion evaluated shortly after reperfusion.[30,31] Thus, a very early MCE assessment of microvascular status may foster the ‘illusion of reperfusion’ (Figure 10.4A–D). The dynamic nature of myocardial perfusion during evolving AMI[32] is further supported by a recent report showing that during infarction early improvement of myocardial contrast perfusion pattern in segments with initially impaired reflow is associated with contractile reserve and with a moderate but significant improvement in regional myocardial function at follow-up.[25] In contrast, segments with sustained no-reflow exhibited no significant contractile reserve or functional recovery. These results suggest that early recovery of microvascular damage is possible and is associated with myocardial viability.

As previously mentioned, these studies involved direct coronary injections of microbubbles in the catheterization laboratory. The availability of intravenous agents and the parallel technical advances in echocardiographic imaging show much promise for the noninvasive exploration of the ischemic and reperfused myocardium on a patient-by-patient basis. This approach has the potential to increase the clinical applications of MCE, making information on perfusion available to a large population of patients in different settings. However, many questions about intravenous MCE await answers

involving methodological, interpretative, and training issues, making the technique still an investigative one.[33]

References

1. Pfeffer MA, Braunwald E. Ventricular remodeling after myocardial infarction: experimental observations and clinical implication. Circulation 1990; 81:1161–72.
2. Ito H, Tomooka T, Sakai N, et al. Lack of myocardial perfusion immediately after successful thrombolysis. A predictor of poor recovery of left ventricular function in anterior myocardial infarction. Circulation, 1992; 85:1699–705.
3. Anselmi M, Bolognese L, Chierchia S, Maggioni A, Marino P. The role of myocardial viability in deriving benefit from reestablishing infarct-related artery flow after acute myocardial infarction. Prog Cardiovasc Dis 2000; 42:455–70.
4. Ito H, Maruyama A, Iwakura K, et al. Clinical implications of the 'no reflow' phenomenon: a predictor of complications and left ventricular remodeling in reperfused myocardial infarction. Circulation 1996; 93:223–8.
5. Kassab GS, Lin DH, Fung YB. Morphometry of pig coronary venous system. Am J Physiol 1994; 267:H2100–13.
6. Kloner RA, Ganote CE, Jennings RB. The 'no-reflow' phenomenon after temporary coronary occlusion in the dog. J Clin Invest 1974; 54:1496–508.
7. Johnson WB, Malone SA, Pantley GA, et al. No reflow and extent of infarction during maximal vasodilation in the porcine heart. Circulation 1988; 78:462–72.
8. Ragosta M, Camarano G, Kaul S, Powers ER, Sarembock IJ, Gimple LW. Microvascular integrity indicates myocellular viability in patients with recent myocardial infarction. New insights using myocardial contrast echocardiography. Circulation 1994; 89:2562–9.
9. Balcells E, Powers ER, Lepper W, et al. Detection of myocardial viability by contrast echocardiography in acute infarction predicts recovery of resting function and contractile reserve. J Am Coll Cardiol 2003; 41:827–33.
10. Lepper W, Kamp O, Vanoverschelde JL, et al. Intravenous myocardial contrast echocardiography predicts left ventricular remodeling in patients with acute myocardial infarction. J Am Soc Echocardiogr 2002; 15:849-56.
11. Ito H, Tomooka T, Sakai N, et al. Time course of functional improvement in stunned myocardium in risk area in patients with reperfused anterior infarction. Circulation 1993; 87:335-62.
12. Agati L, Voci P, Bilotta F, et al. Influence of residual perfusion within the infarct zone on the natural history of left ventricular dysfunction after acute myocardial infarction: a myocardial contrast echocardiographic study. J Am Coll Cardiol 1994; 24:336-42.
13. Camarano G, Ragosta M, Gimple LW, Powers ER, Kaul S. Identification of viable myocardium with contrast echocardiography in patients with poor left ventricular systolic function caused by recent or remote myocardial infarction. Am J Cardiol 1995; 75:215-19.
14. Kenner MD, Zajac EJ, Kondos GT, et al. Ability of the no-reflow phenomenon during acute myocardial infarction to predict left ventricular dysfunction at one-month follow-up. Am J Cardiol 1995; 76:861-8.
15. Czitrom D, Karila-Cohen D, Brochet E, et al. Acute assessment of microvascular perfusion patterns by myocardial contrast echocardiography during myocardial infarction: relation to timing and extent of functional recovery. Heart 1999; 81:12-16.
16. Bolognese L, Parodi G, Carrabba N, et al. Impact of microvascular dysfunction on left ventricular remodeling and long-term clinical outcome after primary coronary angioplasty for acute myocardial infarction. Circulation 2004; 109:1121-6.

17. Colonna P, Cadeddu C, Montisci R, et al. Post-infarction microvascular integrity predicts myocardial viability and left ventricular remodeling after primary coronary angioplasty. A study performed with intravenous myocardial contrast echocardiography. Ital Heart J 2002; 3:506-13.
18. Garot P, Pascal O, Simon M, et al. Impact of microvascular integrity and local viability on left ventricular remodeling after reperfused acute myocardial infarction. Heart 2003; 89:393-7.
19. Sakuma T, Hayashi Y, Sumii K, Imazu M, Yamakido M. Prediction of short- and intermediate-term prognoses of patients with acute myocardial infarction using myocardial contrast echocardiography one day after recanalization. J Am Coll Cardiol 1998; 32:890-7.
20. White FC, Sanders M, Bloor CM. Regional redistribution of myocardial blood flow after coronary occlusion and reperfusion in the conscious dog. Am J Cardiol 1978; 42:234-43.
21. Kaul S, Pandian NG, Guerrero JL, Gillam LD, Okada RD, Weyman AE. The effects of selectively altering the collateral driving pressure on regional perfusion and function in the occluded coronary bed in the dog. Circ Res 1987; 61:77-85.
22. Villanueva FS, Glasheen WP, Sklenar J, Kaul S. Characterization of spatial patterns of flow within the reperfused myocardium by myocardial contrast echocardiography. Implications in determining the extent of myocardial salvage. Circulation 1993; 88:2596-606.
23. Bolognese L, Antoniucci D, Rovai D, et al. Myocardial contrast echocardiography versus dobutamine echocardiography for predicting functional recovery after acute myocardial infarction treated with primary coronary angioplasty. J Am Coll Cardiol 1996; 28:1677-83.
24. Iliceto S, Galiuto L, Marchese A, et al. Analysis of microvascular integrity, contractile reserve, and myocardial viability after acute myocardial infarction by dobutamine echocardiography and myocardial contrast echocardiography. Am J Cardiol 1996; 77:441-5.
25. Brochet E, Czitrom D, Karila-Cohen D, et al. Early changes in myocardial perfusion patterns after myocardial infarction: relation with contractile reserve and functional recovery. J Am Coll Cardiol 1998; 32:2011-17.
26. Agati L, Voci P, Autore C, et al. Combined use of dobutamine echocardiography and myocardial contrast echocardiography in predicting regional dysfunction recovery after coronary revascularization in patients with recent myocardial infarction. Eur Heart J 1997; 18:771-9.
27. Sciagrà R, Bolognese L, Rovai D, et al. Detecting myocardial salvage after primary PTCA: early myocardial contrast echocardiography versus delayed Sestamibi perfusion imaging. J Nucl Med 1999; 40:363-70.
28. Bodenheimer MM, Banka VS, Hermann GA, Trout RG, Pasdar H, Helfant R. Reversible asinergy: histopathologic and electrocardiographic correlations in patients with coronary artery disease. Circulation 1976; 53:792-6.
29. Kloner RA, Rude RE, Carlson N, Maroko PR, DeBoer LWV, Braunwald E. Ultrastructural evidence of microvascular damage and myocellular injury after coronary artery occlusion: which comes first? Circulation 1980; 62:945-52.
30. Lim YJ, Nanto S, Masayama T, Kohama A, Hori M, Kamada T. Myocardial salvage: its assessment and prediction by the analysis of serial myocardial contrast echocardiograms in patients with acute myocardial infarction. Am Heart J 1994; 128:649-56.
31. Ito H, Iwakura K, Oh H, et al. Temporal changes in myocardial perfusion patterns in patients with reperfused anterior wall myocardial infarction. Their relation to myocardial viability. Circulation 1995; 91:656-62.
32. Bolognese L, Cerisano G: Perfusion after acute myocardial infarction. Heart Metab 2001; 13:35-8
33. DeMaria A, Cotter B, Ohmori K. Myocardial contrast echocardiography: too much, too soon? (Editorial). J Am Coll Cardiol 1998; 32:1270-1.

11
Echocardiography in cardiogenic shock

Michael H Picard

Key points

- The incidence of cardiogenic shock after myocardial infarction (MI) remains relatively unchanged, and it is still a major cause of death in patients hospitalized with acute ML
- The prudent use of echocardiography can assist in the management of these patients and assist in reducing the high mortality of this disease.
- The role of echo in cardiogenic shock is at least twofold. First is the use of echocardiography early after presentation to assist in the rapid diagnosis of the cause of shock, and second is its use later to assess the response or effect of treatment.
- The most common cause of post-MI cardiogenic shock is severe left ventricular (LV) dysfunction, but other etiologies include mechanical complications or MI and right ventricular (RV) infarction.
- Early assessment of the severity of mitral regurgitation (MR) and the left ventricular ejection fraction (LVEF) are strong predictors of prognosis in patients with cardiogenic shock from LV dysfunction.

Despite tremendous advances in the treatment of myocardial infarction (MI) over the last 25 years, the incidence of cardiogenic shock after MI remains relatively unchanged and it is still a major cause of death in patients hospitalized with acute MI.[1–4] Patients with cardiogenic shock enrolled in the Global Utilization of Streptokinase and Tissue-Plasminogen Activator for Occluded Coronary Arteries (GUSTO-1) trial represent the largest prospectively collected population of post-MI cardiogenic shock. In this trial, cardiogenic shock occurred in 7.2% of MI patients enrolled, and the 30-day mortality rate for shock patients ranged from 57% for those presenting with shock to 55% for those developing shock after enrollment, and this is compared to a 30-day mortality rate of 3% in the trial for those without shock.[5] Likewise, in GUSTO-IIB, the 30-day mortality for shock patients ranged from 63% in the presence of ST elevation to 72.5% for those without ST-segment elevation;[2] in GUSTO-III, it was approximately 64% for all shock patients. In a US single community study of MI from 1975 to 1997, the incidence of shock was found to be about 7.1% of MI patients with an average in-hospital mortality of 71.7%.1,3 Despite some signs that survival may have improved in the latter half of the 1990s, this syndrome remains a challenge. The prudent use of echocardiography can assist in the management of these patients and assist in reducing the high mortality of this disease.

The role of echo in cardiogenic shock is at least twofold. First is the use of echocardiography early after presentation to assist in the rapid diagnosis of the cause of shock, and second is its use later to assess the response or effect of treatment. While

clinical characteristics that predict the development of cardiogenic shock have been determined,[6] there are limited data on early markers that predict outcome in this disease. Although echocardiographic evaluation of cardiac structure and function is widely utilized in MI, assessing its value acutely in cardiogenic shock has been more challenging. It has only recently been established that echocardiography and specific features of cardiac structure or function early in the course of cardiogenic shock can provide prognostic value and assist in triage to appropriate therapy.[7,8]

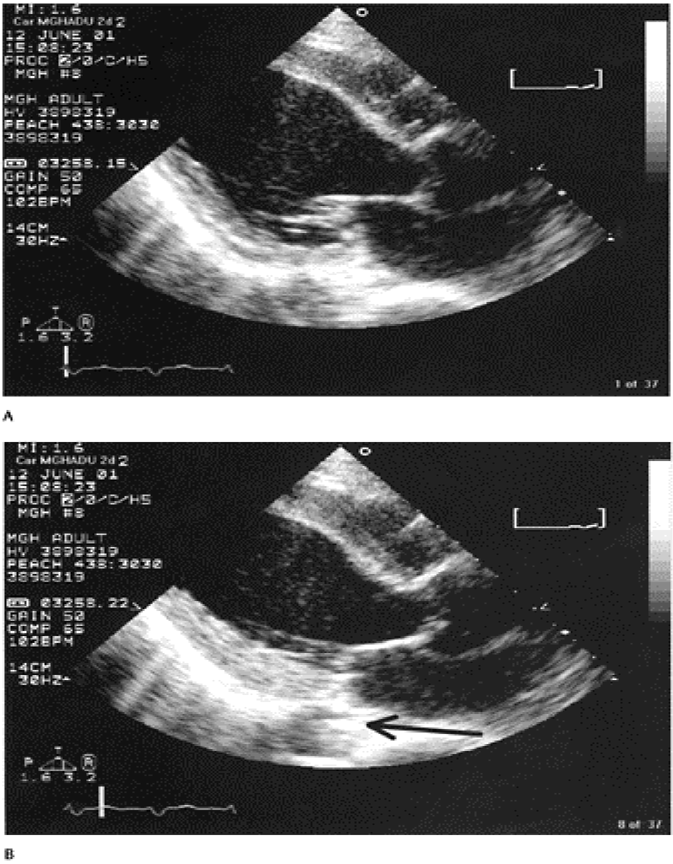


Figure 11.1 Echocardiogram of a patient presenting cardiogenic shock due to extensive LV dysfunction after anterior myocardial infarction. (A) Parasternal long-axis view at end diastole shows a dilated LV. (B) Parasternal long-axis view at end systole shows thickening of only the

basal posterior wall. A bright echo from an intra-aortic balloon pump is noted in the descending thoracic aorta (arrow).

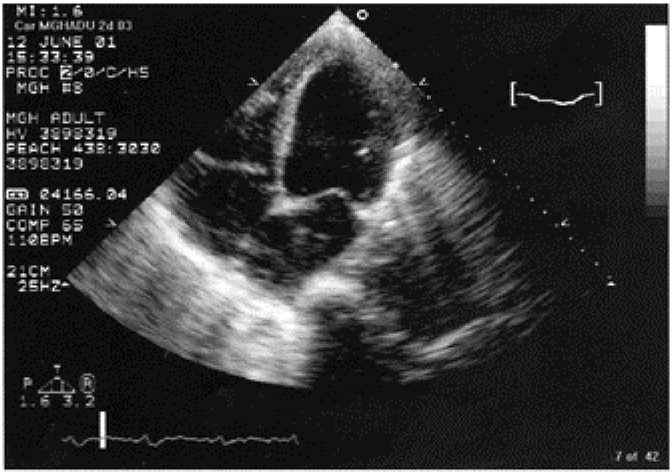
Establishing the etiology of cardiogenic shock

Since transthoracic echocardiography (TTE) provides more information about infarct size and location than electrocardiography, it can be used acutely in the evaluation of the patient with shock or symptoms of heart failure to confirm the presence of left ventricular (LV) wall motion abnormality and thus speed the diagnosis of MI. This is especially helpful when the symptoms are out of proportion to the infarct size on the EGG. Moreover, in the patient with left bundle branch block or other EGG abnormalities that may obscure the classic EGG findings in MI, the echocardiogram can be used to establish the diagnosis of significant LV dysfunction as the cause of shock.

While the most common cause of post-MI cardiogenic shock is severe LV dysfunction from extensive infarction (Figure 11.1), one must also consider other causes of shock. In an international registry of cardiogenic shock, as many as 15% of those presenting with post-MI cardiogenic shock were found to have a cause other than LV dysfunction.[9] Acute mitral regurgitation (MR) or ventricular septal rupture was found in 8%, isolated right ventricular (RV) shock in 2%, and other conditions, including severe valve disease, hypovolemia, or sepsis, in 5%. These data suggest that clinical evaluation alone is not accurate enough to establish the etiology of shock in all post-MI patients. In addition, this registry found that it took a much longer time to make the diagnosis of a mechanical cause of shock. Unfortunately, it is in these patients that a prompt diagnosis is critical, so that correc tive surgery can be instituted prior to the development of irreversible end-organ damage.

The most common echocardiographic findings early in the course of cardiogenic shock are those of a dilated hypocontractile LV (Figure 11.1). In those with prior infarctions, the entire ventricle may show diffuse hypokinesis or akinesis. In those with shock due to an extensive first MI, a more regional pattern of wall motion reflecting the coronary distribution will be present. In those with a significant infarction but patency of one or both of the other coronary arteries, a zone of hyperkinesis may be identified in the early stages of MI. This is a compensatory response to the falling stroke volume. In contrast, if, in cardiogenic shock, this early compensatory hyperkinesis is not noted, there is increased likelihood that significant coronary artery disease exists in the other territories. In addition to LV dysfunction, RV involvement may or may not be present. Studies of heart failure patients suggest that those with biventricular involvement have a poorer outcome.[10,11] MR is commonly present in the enlarged hypokinetic heart. Quantification by color Doppler may be more challenging when the heart rate is increased, limiting the period of systole available for Doppler detection of regurgitation. Moreover, as the pressure difference between the LV and the left atrium (LA) decreases as the LV dP/dt decreases and the LA pressure rises, the MR velocity may be more

difficult to detect. For those in sinus rhythm, a Restrictive’ transmitral velocity filling pattern is often detected by pulse-wave Doppler, reflecting the decreased LV compliance and elevated LA pressures. Numerous studies in heart failure patients have identified this as a marker of adverse outcome.[12]



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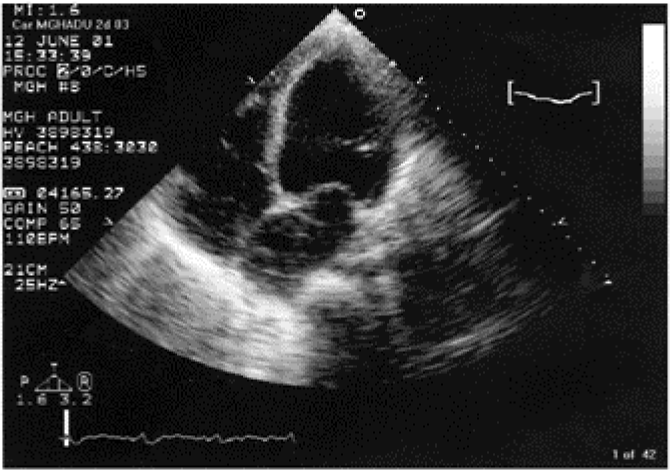


D

(C) Apical four-chamber view at end diastole shows a dilated LV and RV. The interatrial septum is displaced to the right consistent with increased LA volume and pressure. (D) Apical four-chamber view at end systole shows

minimal change in LV area consistent with extensive LV dysfunction and markedly depressed LVEF. Decreased RV systolic function is also present.

As discussed elsewhere in this book, echocardiography, especially TTE, is uniquely suited to make the diagnosis of mechanical complications (Chapter 15). A knowledge of the risk factors for post-MI mechanical complications is important to increase one's level of suspicion of these abnormalities. The risk factors include older age, female gender, first MI, hypertension, delayed recognition of infarction, and continued physical activity after MI.

In the shock patient with a cause other than extensive LV dysfunction to explain the condition, it is not unusual for the LV to be small and hyperkinetic. Thus, even if the image quality is inadequate on TTE to identify a papillary muscle rupture or ventricular septal rupture, the presence of a small, hyperkinetic LV should raise the suspicion of a mechanical complication. Table 11.1 shows the most common abnormalities seen in patients presenting with cardiogenic shock. Each of them has findings on the echocardiogram that should discriminate it from the others.

Figure 11.2 shows an example of the value of early echocardiography in a 72-year-old women who developed cardiogenic shock in the first week after acute MI. TTE revealed a small hyperkinetic LV, which was not expected from the physical examination, which detected tachycar-

Table 11.1 Etiology of shock in the post-MI patient

Extensive LV dysfunction
RV infarction
Papillary muscle rupture
Ventricular septal rupture
LV free-wall rupture with or without pseudoaneurysm formation
Aortic dissection
Sepsis
Hemorrhage and other causes of hypovolemia

dia, pulmonary edema, and hypotension. While no mechanical complication was noted on the TTE, the image quality did not allow a complete evaluation of all aspects of the mitral valve; thus, a transesophageal echocardiogram was performed, and the diagnosis of papillary muscle rupture was made. Prompt triage to the operating room was the optimum treatment in this case.

While TTE is typically the first imaging tool used in the early assessment of cardiogenic shock, transesophageal echocardiography (TEE) can be safely utilized by expert hands. Careful attention to the type and degree of medications used for sedation and their potential for exacerbating the altered vital signs is critical. Important indications for TEE are as follows: (1) when the TTE is of inadequate quality to visualize cardiac structures; (2) any time the clinician has a high index of suspicion of a mechanical complication and it is not identified by TTE; (3) when the degree of hemodynamic instability is unexplained, as in a patient with a small region of abnormal LV wall motion

yet cardiogenic shock, or a patient who remains in shock despite aggressive treatment including reperfusion/revascularization. With a current focus on acute MI treatment strategies that include primary percutaneous coronary interventions that may not include LV angiography, one must remember that such revascularization procedures will not reverse the effects of a papillary muscle transection or ventricular septal rupture that occurred prior to the intervention. Thus, TEE may be a critical adjunct in the patient with persistent hemodynamic compromise despite an open infarct-related artery.

Role of early echocardiography as a prognostic tool

The SHOCK trial is a recently completed international, multicenter, randomized trial of acute treatment in

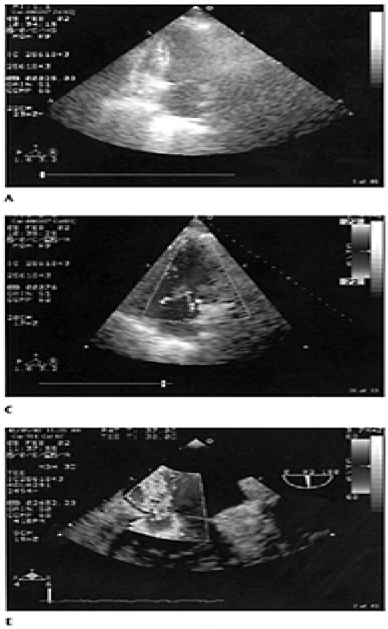


Figure 11.2 Papillary muscle rupture in a 72-year-old woman who developed cardiogenic shock 3 days after acute inferior myocardial infarction. (A and B) Transthoracic apical two-chamber views at end diastole and end systole, respectively, show normal LV size and excellent systolic function. (C) Color Doppler

from the apical view shows an eccentrically directed jet of MR. (D) Transesophageal echo shows the papillary muscle rupture and flail mitral valve leaflets. (E) Color Doppler during the TEE shows severe MR through a large regurgitant orifice.

cardiogenic shock. Cardiogenic shock was strictly defined on both a clinical and a hemodynamic basis. Those with RV infarction or a mechanical complication of MI as an explanation for their shock were prospectively excluded from the trial. Within 48 h of myocardial infarction and 12 h of shock onset, patients were randomized to early revascularization (ERV) or initial medical stabilization (IMS). For the ERV arm, angioplasty or bypass surgery had to be performed within 6 h of randomization; intraaortic balloon counterpulsation was recommended. For patients randomized to the IMS group, intensive medical therapy was required, and intra-aortic balloon counter-pulsation and thrombolytic therapy were recommended. Delayed revascularization at a minimum of 54 h after randomization was recommended if clinically appropriate. The trial demonstrated a 6-month and 1-year survival benefit for patients who received early emergency revascularization compared with initial medical stabilization.[13,14] Two-dimensional echocardiograms were performed on entry into this trial and thus offer a unique opportunity to assess the value of echocardiography and clarify the pathophysiology of this disease.

As is typical of the management cardiogenic shock patients, 85% of the echocardiograms were performed after the patients were stabilized with a combination of intra-aortic balloon pump, dopamine, or norepinephrine but prior to interventions such as revascularization. The mean LVEF of the 175 early echocardiograms available for analysis was 30±11.5%, demonstrating that there is a wide range of systolic function in patients with cardiogenic shock. At least mild MR was found in 39% of the patients, and the mean wall motion score index was 2.1 per segment.[7,8]

The significant univariate predictors of 1-year survival were LV ejection fraction, LV end diastolic volume, LV end systolic volume, and the severity of MR. For example, regardless of the treatment arm, patients with less than mild MR on the entry echocardiogram had a 58% 1-year survival compared to a 31% 1-year survival for those with mild or more MR. The median entry LVEF in the trial was 28%. Those with an LVEF of less than 28% had a 24% 1-year survival compared to a 56% 1-year survival for those with LVEF greater than or equal to 28%. Multivariate analysis showed that both LVEF and MR provided independent prognostic information. Regardless of the treatment arm, an important survival difference was noted for those with 0 to 1+ MR and an LVEF greater than or equal to 28% on presentation with shock (70% 1-year survival) compared to patients with 2+ to 4+ MR and LVEF of less than 28% (10% 1-year survival).[7,8]

An important finding of this study is that the prognostic features on the echocardiogram early in the course of cardiogenic shock (LVEF and MR) are the same as for less complicated MI except that the cutoff point or threshold value may differ.[15–17]

As would be expected, the extent and severity of regional dysfunction, the end diastolic volume, the end systolic volume, and the sphericity of the LV all influenced the LVEF. The degree of MR on this early echocardiogram was related to factors which influenced mitral leaflet geometry, specifically the LV end diastolic volume.[7,8]

A comparison of echocardiograms performed before and after revascularization suggested that LVEF was higher and LV volume lower after the revascularization procedure.[7,8] This observation suggests that the acute revascularization in cardiogenic shock results in improvement of ischemia and a quicker recovery of stunned myocardium.

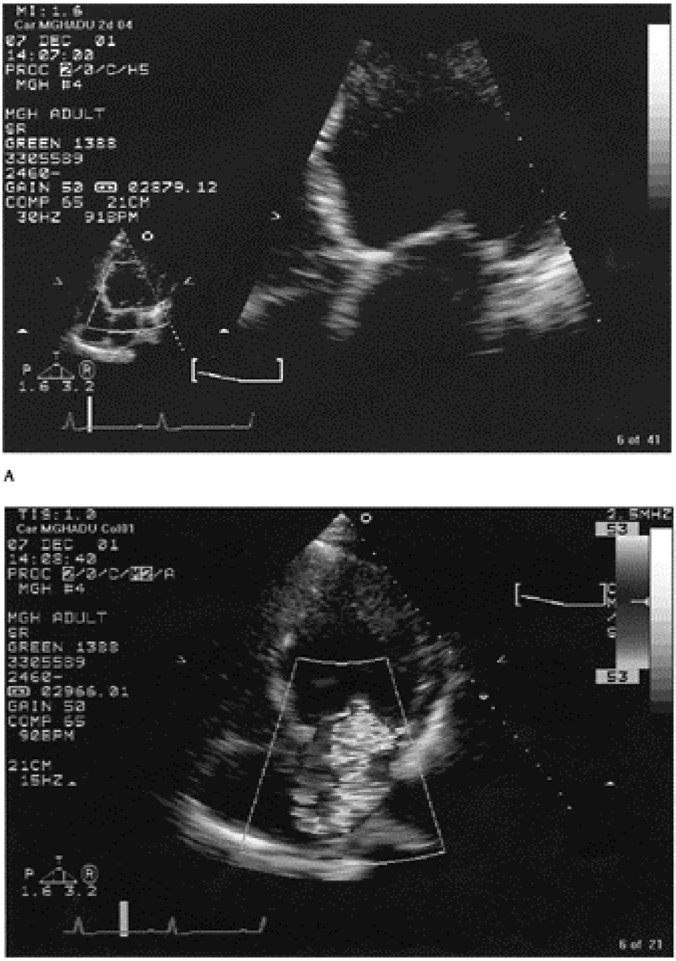
Role of later echocardiography to assess response to therapy

For patients who have recovered from their shock syndrome, echocardiography can be of value later in their hospital course. Firstly, this later assessment of cardiac structure and function can determine whether there has been a significant improvement over presentation. This particularly includes assessment of LVEF, regional LV wall motion (as a marker of infarct size), mitral regurgitation, RV systolic pressure, and markers of LV remodeling such as LV size, volume, and sphericity.[18–20]

This later echocardiogram can assist management in several ways. For example, the presence of decreased LV systolic function and significant regional LV dysfunction in the patient with persistent severe heart failure despite maximal medical therapy may prompt the following: (1) consideration of revascularization in those who have not had such therapy; (2) consideration of a more complete revascularization in those who had undergone an early intervention on a culprit lesion; (3) consideration of long-term mechanical support with LV assist or other devices;[21] (4) consideration of cardiac transplant. On the other hand, the presence of a persistent decrease in functional capacity despite evidence of a significant improvement in LV function on the echocardiogram might provide evidence that the patient’s clinical status is due to other problems such as MR, deconditioning, other valve disease, or concomitant lung disease.

In addition to quantification of MR, identification of the mechanism of MR may be important to determine whether mitral valve (MV) reconstruction surgery is necessary or feasible.[22] Typically, in these patients with dilated LV, a critical part of the interpretation is to distinguish functional MR due to a dilated LV and MV annulus from a structural abnormality of the MV apparatus such as chordal rupture or MV prolapse. The presence of the incomplete MV leaflet closure pattern identifies those patients with MR due to an acutely remodeled LV (Figure 11.3).[23–25]

A careful quantitation of the regional LV wall motion on this later echocardiogram and comparison to one performed earlier in the course of the disease can help identify areas of stunned myocardium which may have recovered. In addition, if there is any question about the suitability of a particular patient for late revascularization, the use of dobutamine echocardiography can identify those patients with viable myocardium and significant coronary artery disease.[26]



A

B

Figure 11.3 Functional MR in a dilated LV. (A) This magnified view of the MV leaflets from an apical four-chamber view during systole shows the incomplete mitral leaflet closure pattern, which in this case is due to the dilated LV. (B) Significant MR is noted on color Doppler. The jet is centrally directed, and a prominent zone of flow convergence is seen on the LV side of the valve.

Summary

Two-dimensional echocardiography has several roles in the patient presenting with cardiogenic shock. First, early echocardiography can assist in the prompt diagnosis of the etiology of the hemodynamic instability, by either confirming or establishing the diagnosis. This can lead to appropriate therapy. Second, the echocardiogram can assist in assessing prognosis. Identification of high- and low-risk groups on the basis of the severity of MR and the LVEF offers the potential for more tailored therapy for individuals early in the course of disease. Lastly, echocardiography later in the disease course can assist in assessing response to therapy and identifying other causes of symptoms that mimic ischemic heart disease.

References

1. Goldberg RJ, Gore JM, Alpert JS, et al. Cardiogenic shock after acute myocardial infarction. Incidence and mortality from a community-wide perspective, 1975 to 1988. N Engl J Med 1991: 325:1117–22.
2. Holmes DR Jr, Bates ER, Kleiman NS, et al. Cardiogenic shock in patients with acute ischemic syndromes with and without ST segment elevation. Circulation 1999; 100:2067–73.
3. Goldberg RJ, Samad NA, Yarzebski J, Gurwitz J, Bigelow C, Gore JM. Temporal trends in cardiogenic shock complicating acute myocardial infarction. N Engl J Med 1999; 340:1162–8.
4. Dauerman HL, Goldberg RJ, Malinski M, Yarzebski J, Lessard D, Gore JM. Outcomes and early revascularization for patients ≥65 years of age with cardiogenic shock. Am J Cardiol 2001; 87:844–8.
5. Holmes DR Jr, Bates ER, Kleiman NS, et al for the GUSTO-I Investigators. Contemporary reperfusion therapy for cardiogenic shock: the GUSTO-I Trial experience. J Am Coll Cardiol 1995; 26:668–74.
6. Hasdai D, Califf RM, Thompson TD, et al. Predictors of cardiogenic shock after thrombolytic therapy for acute myocardial infarction. J Am Coll Cardiol 2000; 35:136–43.
7. Picard MH, Davidoff R, Sleeper LA, et al. Echocardiographic predictors of survival and response to early revascularization in cardiogenic shock. Circulation 2003; 107:279–84.
8. Picard MH, Davidoff R, Mendes LA, et al. Determinants of mortality from cardiogenic shock: observations from the SHOCK trial. Circulation 1999; 100:1–18.
9. Hochman JS, Boland J, Sleeper LA, et al and the SHOCK Registry Investigators. Current spectrum of cardiogenic shock and effect of early revascularization on mortality. Circulation 1995; 91:873–81.
10. Mendes LA, Dec GW, Picard MH, Palacios IF, Newell J, Davidoff R. Right ventricular dysfunction: an independent predictor of outcome in patients with myocarditis. Am Heart J 1994; 128:301–7.
11. Mendes LA, Picard MH, Jacobs AK, et al. Cardiogenic shock: predictors of outcome based on right and left ventricular size. Circulation 1999; 100:1–19.
12. Hansen A, Haass M, Zugck C, et al. Prognostic value of Doppler echocardiographic mitral inflow patterns: implications for risk stratification in patients with chronic congestive heart failure. J Am Coll Cardiol 2001; 37:1049–55.
13. Hochman JS, Sleeper LA, Webb JG, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. N Engl J Med 1999; 341:625–34.
14. Hochman JS, Sleeper LA, White HD, et al. One-year survival following early revascularization for cardiogenic shock. JAMA 2001; 285:190–2.

15. Feinberg MS, Schwammenthal E, Shlizerman A, et al. Prognostic significance of mild mitral regurgitation by color Doppler echocardiography in acute myocardial infarction. Am J Cardiol 2000; 86:903–7.
16. Multicenter Post-infarction Research Group. Risk stratification and survival after myocardial infarction. N Engl J Med 1983; 309:331–6.
17. Volpi A, DeVita C, Franzosi MG, et al. Determinants of 6-month mortality in survivors of myocardial infarction after thrombolysis: results of the GISSI-2 data base. Circulation 1993; 88:416–29.
18. Picard MH, Wilkins GT, Ray PA, Weyman AE. Natural history of left ventricular size and function after acute myocardial infarction. Assessment and prediction by echocardiographic endocardial surface mapping. Circulation 1990; 82:484–94.
19. Picard MH, Wilkins GT, Thomas JD, Gillam LD, Weyman AE. Immediate regional endocardial surface expansion with coronary occlusion in the canine left ventricle: disproportionate effects of anterior vs. inferior ischemia. Am Heart J 1991; 121:753–62.
20. Mendes LA, Picard MH, Dec GW, Hartz VL, Palacios IF, Davidoff R. Ventricular remodeling in active myocarditis. Myocarditis Treatment Trial. Am Heart J 1999; 138:303–8.
21. Rose EA, Gelijns AC, Moskowitz AJ, et al and the Randomized Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure (REMATCH) Study Group. Long-term mechanical left ventricular assistance for end-stage heart failure. N Engl J Med 2001; 345:1490–3.
22. Picard MH. The role of transesophageal echocardiography in selecting the optimum surgical approach for the regurgitant valve: repair vs. replacement. MDConsult Cardiology, 25 Sept 2001.
23. Otsuji Y, Handschumacher MD, Liel-Cohen N, et al. Mechanism of ischemic mitral regurgitation with segmental left ventricular dysfunction: three-dimensional echocardiographic studies in models of acute and chronic progressive regurgitation. J Am Coll Cardiol 2001; 37:641–8.
24. Messas E, Guerrero JL, Handschumacher MD, et al. Paradoxical decrease in ischemic mitral regurgitation with papillary muscle dysfunction: insights from three dimensional and contrast echocardiography with strain rate measurement. Circulation 2001; 104:1952–7.
25. Kaul S, Spotnitz WD, Glasheen WP, Touchstone DA. Mechanism of ischemic mitral regurgitation. An experimental evaluation. Circulation 1991; 84:2167–80.
26. Nijland F, Kamp O, Verhorst PM, deVoogt WG, Visser CA. In-hospital and long-term prognostic value of viable myocardium detected by dobutamine echocardiography early after acute myocardial infarction and its relation to indicators of left ventricular systolic dysfunction. Am J Cardiol 2001; 88:949–55.

12
Echocardiography in acute aortic dissection

Frank A Flachskampf

Key points

- The most important clue to diagnosis of aortic dissection is to keep its possibility in mind in all patients with chest pain or shock.
- The presence of (new) aortic regurgitation or pericardial effusion on transthoracic echo in a patient with chest pain is highly suggestive of type A dissection, even if a dissection flap is not immediately detectable.
- A significant blood pressure rise due to discomfort or gagging during transesophageal echocardiography (TEE) must be avoided.
- While diagnosis of dissection in the descending aorta is mostly easy, both false-positive and false-negative findings occur in the ascending aorta. Use additional computed tomography or magnetic resonance imaging if the diagnosis is not evident.

Clinical and pathophysiologic background

Aortic dissection occurs when intramural aortic hemorrhage or bleeding into the aortic wall leads to separation of the aortic intima from the adventitia, creating the typical dissection membrane or flap. It is more frequent in men than in women. The most frequently associated diseases are arterial hypertension, Marfan’s syndrome, and chest trauma, but aortic dissection can occur spontaneously in nonhypertensive patients. An association with late pregnancy has also been noted.

The ascending aorta is affected in 60% of cases, and the thoracic descending aorta immediately distal to the takeoff of the left subclavian artery in 30% of cases. Based on the segments of the aorta involved in the dissection, two classifications are commonly accepted, Stanford and DeBakey (Table 12.1).

Ascending aortic dissection is an extraordinarily lethal disease, with a mortality rate of up to 1–2% *per hour* in the first 48 h if untreated. Death ensues by aortic rupture into the mediastinum or into the pericardial space. Other severe complications are torrential aortic regurgitation and propagation of dissection into the carotids with subsequent stroke, or into the coronaries with subsequent myocardial infarction. Dissection of the ascending aorta (that is, DeBakey I or II/Stanford A) therefore mandates immediate surgery, and its rapid diagnosis is of the utmost importance. Elevated blood pressure should be lowered and intravenous beta-blockers administered, if blood pressure allows this, to reduce the rate of aortic pressure rise in systole.

In comparison, descending aortic dissection carries a lower risk, mainly of rupture or of organ ischemia, such as renal or enteral ischemia. Dissection of the descending aorta is therefore treated medically by blood pressure control and supportive measures, with surgical standby in case of organ damage.

Table 12.1 Classification of aortic dissection

	Ascending aorta	*Aortic arch*	*Descending aorta*
DeBakey I	Yes	Yes	Yes
DeBakey II	Yes	No	No
DeBakey III	No	No	Yes
Stanford A	Yes	Possible	Possible
Stanford B	No	No	Yes

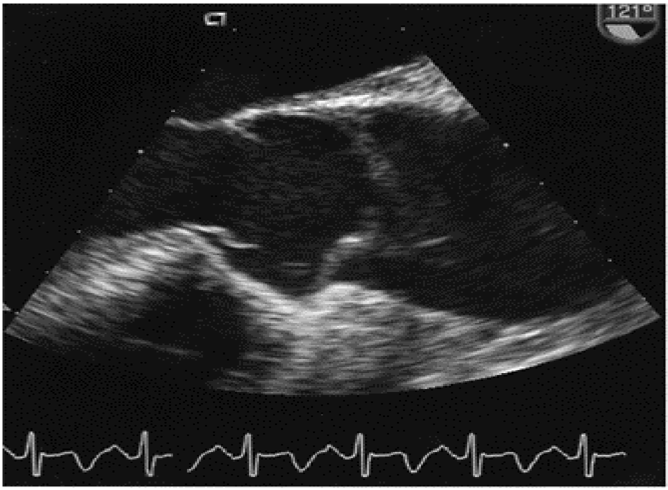


Figure 12.1 Dissection of the ascending aorta seen in a transesophageal long axis-view at 121° in systole. Note mobile flap changing configuration during the cardiac cycle (compare Figure 12.2), and aortic dilation. (Reproduced from Flachskampf and Daniel. Cardiology Clinics 2000; 18:807–17.[2] © 2000 with permission from Elsevier.)

Recently, a new classification of aortic dissection has been proposed which encompasses intramural hematoma and classic dissection.[1] This classification is as follows:

- *class I:* classic dissection of all Stanford or DeBakey types
- *class II:* intramural hematoma
- *class III:* subtle circumscript dissection representing a localized tear without clear-cut hematoma
- *class IV:* plaque ulceration (mostly in the descending aorta, and often in the abdominal aorta)
- *class V:* traumatic or iatrogenic (mostly catheter-induced, retrograde) dissection.

Importantly, dissections of classes II–IV may progress to classic (class I) dissection and are difficult or even sometimes impossible to visualize by transesophageal echocardiography (TEE).

Aortic dissection manifests itself predominantly by severe chest pain, which often changes localization during propagation of the dissection. Other signs include dyspnea due to aortic regurgitation or intrapericardial bleeding, organ or limb ischemia, side difference of arterial pulses, syncope, and shock. Given the enormous morbidity and mortality, especially of ascending aortic dissection, time is of the essence in diagnosis. Unfortunately, the main problem with the diagnosis of aortic dissection is the low index of suspicion. Once the possibility is kept in mind, it is usually not very difficult to establish the diagnosis.

Echocardiographic signs of acute aortic dissection

Features of the dissected aorta

The classic and pathognomonic sign of aortic dissection is the dissection membrane or flap, a thin mobile membrane attached to the wall of the aorta, which separates the true from the false lumen.[2] The membrane is very pliable and changes its shape with the pulse wave (Figures 12.1 and 12.2). Blood pressure and velocity in the true lumen are higher than in the false lumen, and therefore the flap has a convexity toward the false lumen during systole. Most frequently, the false lumen is larger than the true lumen. If the blood velocity in the false lumen is low, spontaneous echo contrast or thrombosis

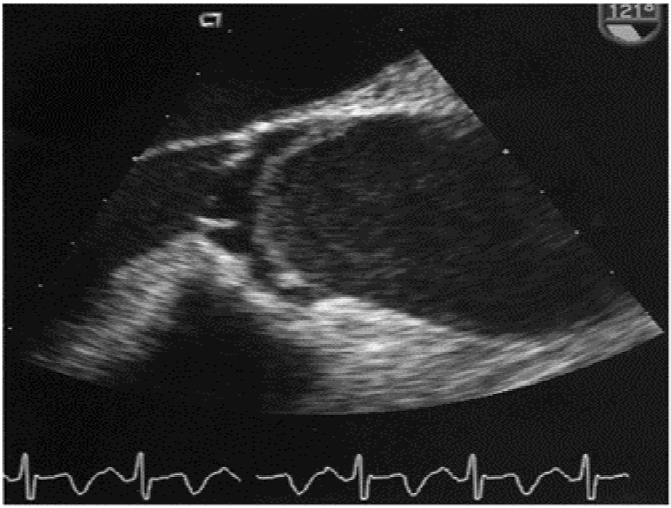


Figure 12.2 Same patient and view as in Figure 12.1 in diastole. (Reproduced from Flachskampf and Daniel. Cardiology Clinics 2000; 18:807–17.[2] © 2000 with permission from Elsevier.)

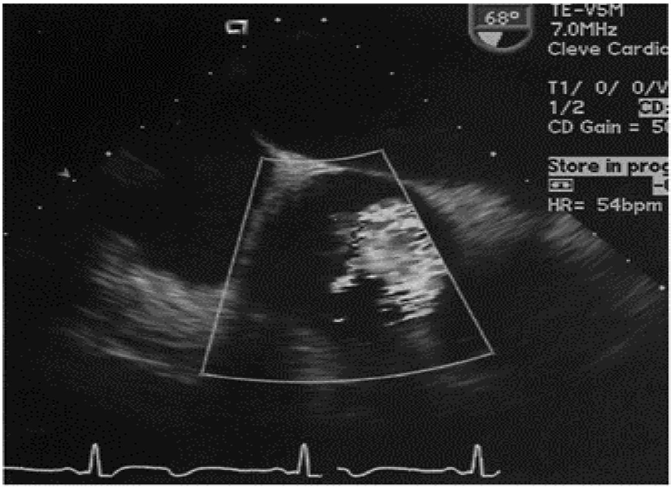


Figure 12.3 Dissection of the ascending aorta in a transesophageal short-axis view at 68°. Note flap and

differentiation of true lumen, with color-coded flow, from surrounding larger false lumen, without visible flow on color Doppler.

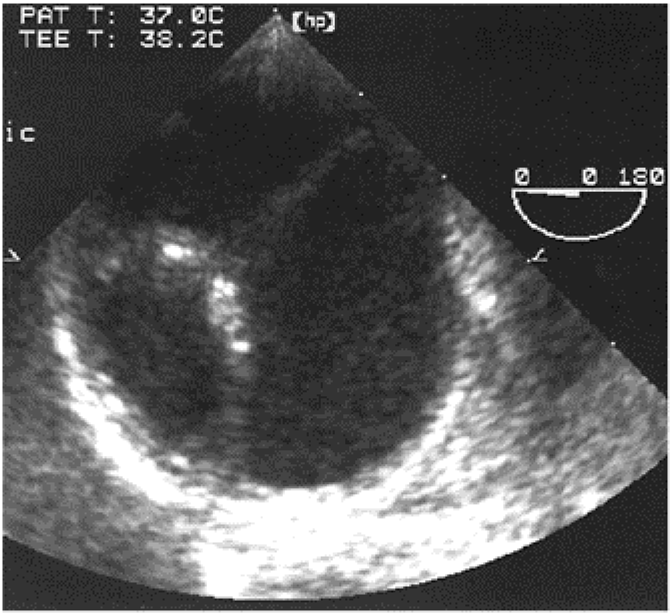


Figure 12.4 Transesophageal short-axis view of a dissection of the descending thoracic aorta. Spider-web-like appearance of the dissection membrane.

may occur. Frequently, by color Doppler, no or only sparse flow signals are seen in the false lumen, while there is color-coded systolic flow in the true lumen (Figures 12.3 and 12.4). This may help during the examination to identify an intraluminal structure as a dissection membrane. The communication sites between true and false lumen are often multiple and are designated entry and re-entry sites, with pulsatile flow from one lumen to the other, visualizable by color Doppler. Typically, entry sites show systolic flow from the true lumen into the false lumen, followed by reverse flow in diastole. Some

Table 12.2 Cardiac abnormalities associated with or suggestive of aortic dissection

Bicuspid aortic valve
Hypertensive heart disease
Hypertensive aortic dilation
Marfan's syndrome (aortic dilation with effacement of the sinotubular junction ridge, central aortic regurgitation, and mitral valve prolapse)
Pericardial effusion
Aortic regurgitation
Inferior hypo/akinesia

authors, on the basis of cases with no apparent entry and no false lumen flow, describe 'noncommunicating dissection' as a distinct disease type, similar to but more extensive than intramural hematoma.

The diameter of the aorta is often, but not necessarily, increased over 4 cm. Periaortic echolucent areas represent either free mediastinal fluid or pleural fluid.

The heart in acute ascending aortic dissection

The following three cardiac structures or events may be acutely affected by dissection of the ascending aorta (Table 12.2):

1. The aortic valve. Very frequently, aortic regurgitation accompanies dissection. The following mechanisms may lead to regurgitation:[3]
   - presence of a bicuspid aortic valve, which is associated with an increased risk of aortic dissection
   - aortic root dilation with central aortic regurgitation
   - interference of the dissection flap with diastolic closure of the aortic cusps. The flap prolapses into the left ventricle in diastole, leading to massive regurgitation. In this case, the valve itself is not damaged and does not necessarily have to be replaced at surgery.
2. Pericardial effusion. This represents bleeding into the pericardial space. It is frequent in dissection of the ascending aorta, and fatal hemorrhage with pericardial tamponade is a frequent immediate cause of death in aortic dissection.
3. Obstruction or dissection of coronary vessels, mostly the right coronary artery, with consequent myocardial ischemia.

Goals and strategy of echocardiography in aortic dissection

Only a few segments of the thoracic aorta are seen on transthoracic echo. The first few centimeters of the ascending aorta are usually seen in the parasternal or apical long-axis view and in the parasternal cranial short-axis view. The descending aorta is seen in cross-section in the parasternal long-axis view and the apical views close to the left atrium. The

images of the aorta in these views often are of limited quality, although in most cases of dissection considerable dilation of the aorta is detected, and often the flap can be seen. However, the presence of more than minimal aortic regurgitation and/or of a pericardial effusion in a patient with chest pain or other suggestive symptoms outlined should immediately suggest aortic dissection to the examiner. In most cases, TEE should be performed to delineate the type and complications of dissection or to exclude the disease altogether.[2,4] In rare cases with excellent image quality and clear visibility of aortic valve, aortic root, and the dissection flap by transthoracic echo, the patient may be sent to surgery without TEE, which still can be performed in the operating room if new questions arise. TEE, because of its discomfort, necessitates vigorous control of blood pressure by sedation and/or antihypertensive medication; cases of aortic rupture during TEE in aortic dissection have been described.[5] Food intake within the last hours is *not* an absolute contraindication to TEE in this particular circumstance; vomiting and aspiration, however, should be anticipated, and in unstable patients tracheal intubation may be wise before TEE.

In the setting of acute aortic dissection, echocardiography must answer the following questions:

1. Presence and extent of aortic dissection; in particular, is the ascending aorta involved? This is the most critical question, since it determines whether the patient should be sent to surgery immediately or not.
2. Is the aortic annulus massively dilated, is there severe aortic regurgitation, and is the aortic valve bicuspid (there is an association between bicuspid aortic valve and aortic dissection)? The answers to these questions, as well as whether the dissection begins in the aortic root or higher, may modify the surgical strategy with regard to aortic valve replacement.
3. Is the dissection membrane in close proximity to the coronary ostia? The right coronary ostium especially may be blocked by the membrane, or the dissection may propagate into the right coronary artery.

Other questions are of interest, but not of vital importance, and answering them should not prolong the examination unduly. The location and configuration of true and false lumen, as well as entry and re-entry sites, usually are not critical for surgery. However, in a few cases, the entry may be more distal than the beginning of the flap. This would suggest retrograde dissection, which may influence surgical strategy in order to resect the primary entry tear.[1] A large pericardial effusion predicts imminent tamponade by free rupture into the pericardial space, hence adding to the urgency. The same is true of the detection of periaortic fluid. The supra-aortic vessels should, in any circumstance, be inspected intraoperatively for dissection; there is little value in going to great efforts to attempt to visualize them by TEE.

TEE in aortic dissection

By TEE, which is best performed with a multiplane probe, the ascending aorta can be seen almost completely in the transesophageal long-axis view of the left ventricle at approximately 120–150° (Figures 12.1 and 12.2), by pulling the instrument gently up

from the lower esophageal position used for the ventricles and mitral valve to a higher transesophageal position.[5] The corresponding ascending aorta short-axis views, at approximately 30–70° (Figure 12.3), after withdrawing the instrument from an aortic valve short-axis view, are important to confirm visualization of structures in the long-axis view, but one should be aware that, because of tracheal and left main bronchus interference, the short-axis views do not reach as far upward toward the aortic arch as the long-axis view does. The short-axis view a few millimeters cranial of the aortic leaflets shows the left main ostium at approximately 2–3 o'clock; the right coronary ostium, at approximately 6 o'clock is less well seen, and often better found by going to a long-axis view, where the takeoff of the right coronary artery away from the transducer can regularly be seen. In elderly patients, immediately distal to the ostium of the right coronary artery, there is frequently an atherosclerotic plaque of the aortic wall, which is a predilection site for the attachment of the dissection membrane.

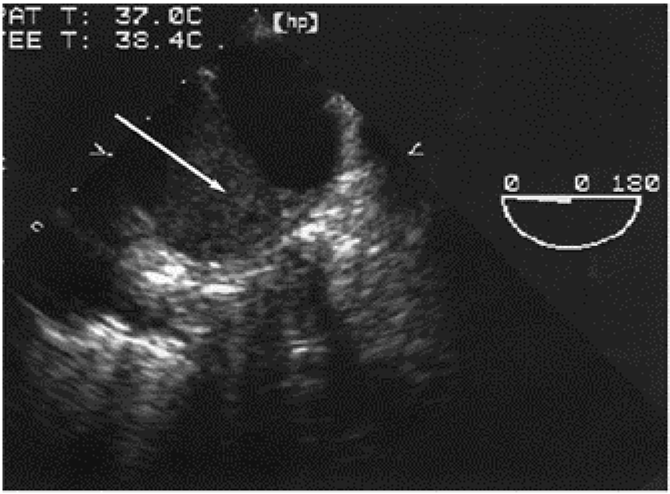


Figure 12.5 Transesophageal short-axis view of a dissection of the descending thoracic aorta. The false lumen (arrow) is thrombosed, showing a higher echodensity than the true lumen. Note dilation of the descending aorta.

Apart from visualizing the dissection membrane as unequivocally as possible, one should note the morphology of the aortic valve, the mechanism of regurgitation, the pericardial effusion, and the thickness of the aortic walls. A rapid look at left and right ventricular function and the mitral valve should also be part of the examination. For examination of the descending aorta, the probe should be positioned as distal as the thoracic descending aorta can be visualized, and then slowly pulled back, preferentially in the 0° horizontal

plane, using other planes for confirmation if pathologic structures are seen. Aortic diameter, presence of a flap, entry/re-entry sites, wall thickening suggesting intramural hematoma, and periaortic fluid should be noted (Figures 12.4 and 12.5). An increase in distance between probe (esophagus) and aorta may indicate intramural or periaortic hematoma. At the takeoff of the left subclavian artery, the distal arch is visualized. By slow retraction of the probe, most of the arch can be inspected, with the anterior/superior aortic wall close to the transducer and the inner curvature of the arch away from the transducer. Involvement of the carotids in a dissection can be much better ascertained by direct transcutaneous ultrasound examination of the carotids than by TEE.

Pitfalls and problems

The dissection membrane usually is unmistakable and can be brought into view very clearly by TEE, separating a zone of high and one of low flow by color Doppler. However, the distal ascending aorta and parts of the aortic arch frequently escape visualization by TEE. In rare cases, type II dissections may hide in this region, although most type I and II dissections begin in or close to the aortic root. False-positive findings of intra-aortic structures also occur more frequently in the ascending aorta, due to reverberation and other artifacts, such as Swan-Ganz catheters.[6, 7] Structures and color-coded flow at exactly double the distance from the transducer of other structures and flows, moving in parallel with each other, should be suspected of being reverberation artifacts. In the descending aorta, reverberation can create the image of a ‘double-barrel’ aorta, with color Doppler flow signals in a ‘second’ aorta distal and parallel to the true aorta.

No matter how experienced the examiner is, not every diagnosis of aortic dissection or intramural hematoma (see below) can confidently be made by TEE alone, and computed tomography or magnetic resonance imaging should be performed if serious doubts remain.

Differential diagnosis

The similarity in presentation to myocardial infarction, which typically is treated by fibrinolysis or percutaneous coronary intervention, warrants an aggressive effort to confirm or refute the diagnosis, since both therapies are contraindicated in dissection, and surgical repair is urgent in ascending aortic dissection. The problem is compounded by the fact that ascending aortic dissection itself may lead to myocardial ischemia (mainly by occluding the right coronary artery). In this case, misdiagnosis of aortic dissection as (primary) myocardial ischemia is almost the rule.

In practice, in the patient with severe chest pain, unclear shock, or unclear neurologic deficit, dissection should be ruled out by TEE if there is a reasonable suspicion that dissection might be the underlying cause. Such suspicion is based on the following symptoms or signs: chest pain changing its localization; asymmetric pulses, neurologic deficit, limb or organ ischemia; mediastinal widening on chest radiograph; pericardial effusion, more than minimal aortic regurgitation, or ascending aortic dilatation on echo.

These signs should suggest dissection even if there is ST-elevation in the EGG, a wall motion abnormality on echo, or a positive troponin test, because myocardial ischemia and aortic dissection are not mutually exclusive.

Severe pulmonary embolism in rare cases may enter the differential diagnosis. Usually, pulmonary embolism causes far less pain and far more dyspnea than dissection does. The echo quickly reveals whether there is striking right ventricular enlargement and hypokinesia, which would strongly argue against aortic dissection.

Intramural hematoma

Intramural hematoma is a variant and an early form of aortic dissection, often coexisting with or progressing to typical dissection.[8–10] Therapeutically, it is managed in the same way as classic dissection, depending on involvement of the ascending aorta. Pericardial effusion may be present. Intramural hematoma is more frequent (or more frequently detected) in the descending than in the ascending aorta. The diagnostic criteria are an aortic wall thickness over 7 mm and/or an echolucent zone in the aortic wall. Unfortunately, an echolucent zone is not always present, and considerable wall thickening may occur due to atherosclerosis itself. Hence, the diagnosis of pure intramural hematoma is not easy, and magnetic resonance imaging or computed tomograpy should be considered in cases of suspicious aortic wall thickening.[11]

Is echo enough to send the patient to surgery for aortic dissection?

As always in echocardiography, the diagnostic quality is decisively influenced by the skills of the examiner. Several comparative studies, however, have clearly shown over the last decades[4, 12, 13] that, in experienced hands, TEE is considerably superior to transthoracic imaging in the diagnosis of aortic dissection, and TEE is practically as good as computed tomography or magnetic resonance imaging for detection of dissection. Hence, TEE in most experienced centers is the first-line approach to the patient with suspected dissection, mainly due to bedside availability and rapidity. However, as already pointed out, there are cases where this technique is not conclusive and another modality should be sought.

References

1. Erbel R, Alfonso F, Boileau C, et al. Diagnosis and management of aortic dissection. Recommendations of the Task Force on Aortic Dissection, European Society of Cardiology. Eur Heart J 2001; 22:1642–81.
2. Flachskampf FA, Daniel WG. Aortic dissection. Cardiol Clin 2000; 18:807–17.
3. Movsowitz HD, Levine RA, Hilgenberg AD, Isselbacher EM. Transesophageal echocardiographic description of the mechanisms of aortic regurgitation in acute type A aortic dissection: implications for aortic valve repair. J Am Coll Cardiol 2000; 36:884–90.

4. Erbel R, Engberding G, Daniel W, Roelandt J, Visser C, Rennollet H, and the European Cooperative Study Group for Echocardiography. Echocardiography in diagnosis of aortic dissection. Lancet 1989; 1:457–61.
5. Flachskampf FA, Decoodt P, Fraser AG, Daniel WG, Roelandt JRTC. Recommendations for performing transesophageal echocardiography. Eur J Echocardiogr 2001; 2:8–21.
6. Appelbe AF, Walker PG, Yeoh JK, Bonitatibus A, Yoganathan AP, Martin RP. Clinical significance and origin of artifacts in transesophageal echocardiography of the thoracic aorta. J Am Coll Cardiol 1993; 21:754–60.
7. Bansal RC, Chandrasekaran K, Ayala K, Smith DC. Frequency and explanation of false negative diagnosis of aortic dissection by aortography and transesophageal echocardiography. J Am Coll Cardiol 1995; 25:1393–401.
8. Mohr-Kahaly S, Erbel R, Kearney P, Puth M, Meyer J. Aortic intramural hemorrhage visualized by transesophageal echocardiography: findings and prognostic implications. J Am Coll Cardiol 1994; 23:658–64.
9. Nienaber CA, von Kodolitsch Y, Petersen B, et al. Intramural hemorrhage of the thoracic aorta. Diagnostic and therapeutic implications. Circulation 1995; 92:1465–72.
10. Harris KM, Braverman AC, Gutierrez FR, Barzilai B, Dàvila-Román VG. Transesophageal echocardiographic and clinical features of aortic intramural hematoma. J Thorac Cardiovasc Surg 1997; 114:619–26.
11. Flachskampf FA, Banbury M, Smedira N, Thomas JD, Garcia, M. TEE diagnosis of intramural hematoma of the ascending aorta: a word of caution. J Am Soc Echocardiogr 1999; 12:866–70.
12. Nienaber CA, Spielmann RP, Kodolitsch YV, et al. Diagnosis of thoracic aortic dissection: magnetic resonance imaging versus transesophageal echocardiography. Circulation 1992; 85:434–47.
13. Nienaber CA, von Kodolitsch Y, Nicolas V, et al. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. New Engl J Med 1993; 328:1–9.

13
Echocardiography in acute mitral regurgitation

Frank A Flachskampf

Key points

- The presence of a clear structural abnormality of the mitral valve on two-dimensional (2-D) echo (such as a flail leaflet with the tip of the leaflet appearing in the left atrium during systole) predicts severe mitral regurgitation, even if the color Doppler appearance is ambiguous. Conversely, the absence of any identifiable morphologic abnormality makes severe regurgitation unlikely.
- A well-formed, consistently imaged proximal acceleration zone on the ventricular side of a mitral regurgitant lesion predicts severe regurgitation.
- Systolic pulmonary venous flow reversal predicts severe regurgitation, but it is not present in all cases of severe regurgitation (high specificity, modest sensitivity).
- Definitive evaluation of regurgitation of a prosthetic mitral valve usually requires transesophageal imaging.

Clinical and pathophysiologic background

Acute severe mitral regurgitation is a cardiovascular emergency encountered in several typical scenarios (Table 13.1). Due to systolic regurgitation into the left atrium, there is an acute volume overload, and, consequently, pressure overload of the left atrium, leading to an acute increase in pulmonary capillary pressure. This is reflected in the typical high systolic V wave in the pulmonary wedge pressure tracing, exceeding double the value of the mean wedge pressure. The pressure increase leads to acute pulmonary edema and, propagating backward or ‘upstream’, to acute pulmonary hypertension and right heart failure. Acute mitral regurgitation can be conceptually understood primarily as left ventricular backward failure, leading secondarily to tachycardia, hypotension, forward failure, and cardiogenic shock. Characteristically, the left ventricle is normal sized and left ventricular global function is hyperkinetic, unless there is additional left ventricular disease. This contrasts with chronic mitral regurgitation, which leads to progressive left ventricular dilation and systolic dysfunction.

Typical clinical signs of mitral regurgitation include a holosystolic murmur over the apex and axilla. However, in the setting of cardiogenic shock and severe pulmonary edema due to acute mitral regurgitation, the murmur may

Table 13.1 Etiology of acute mitral regurgitation

Infective endocarditis with valvular destruction
Papillary muscle rupture following myocardial infarction
Degenerative chordal rupture, particularly in mitral valve prolapse
Mitral prosthetic dysfunction – bioprosthetic degeneration – bioprosthetic endocarditis with leaflet destruction – ring abscess with large paraprosthetic leak or prosthetic dehiscence – prosthetic thrombosis with fixed position of occluder – fracture of prosthetic valve with occluder embolization
Rare causes: trauma, postoperative suture dehiscence, post-valvotomy regurgitation, and others

be barely audible, and its loudness is not predictive of the severity of regurgitation. Rapidly progressive ('flash') pulmonary edema, tachycardia, and forward failure with cardiogenic shock are hallmarks of the condition. Even in the absence of a loud murmur, these signs together with one of the above cited four typical scenarios are strongly suggestive of acute mitral regurgitation. The diagnostic tool of choice is echocardiography, which should be performed quickly and conclusively, proceeding to transesophageal echocardiography (TEE) if any doubts remain after transthoracic imaging. Severe acute mitral regurgitation is not tolerated more than some hours at best. The definitive therapy is surgery, consisting of repair or replacement of the regurgitant valve or prosthesis. Before surgical correction, supportive measures include diuretics, mechanical ventilation with positive end-expiratory pressure for pulmonary edema, and lowering blood pressure to reduce afterload (with nitroprusside or another rapidly acting drug), unless the patient is already hypotensive. An excellent bridge to surgery is intra-aortic balloon counterpulsation, which decreases systolic afterload and increases diastolic systemic pressure. If feasible, it should be instituted once the diagnosis of severe acute mitral regurgitation is clear, possibly before cardiogenic shock ensues. The correct position of the intra-aortic balloon can be ascertained by TEE.

Goals of echocardiography in acute mitral regurgitation

Severe acute mitral regurgitation is usually not difficult to recognize, since there typically is both a large jet of mitral regurgitation and also 2-D evidence that the valve is grossly abnormal. The emergency echo examination of the mitral valve should attempt to answer the following essential questions:

- Is there severe mitral regurgitation?
- Do the mitral valve leaflets, the chordae, and the papillary muscles look normal?
- Is there leaflet thickening?
- Are there vegetations?
- Is there excessive mobility of leaflets or other parts of the mitral apparatus, leading to erratic motion, prolapse, or flail during systole? By TEE, the location of a prolapse or flail with the origin of the regurgitant jet can often be localized well in the transgastric,

short-axis view of the left ventricle, or else by carefully scanning the whole mitral valve from a low trans—esophageal, four-chamber view position with stepwise increments in cross-section angle. A break in the continuity of the subvalvular apparatus, as in chordal or papillary muscle rupture, is often best visualized in the transgastric, two-chamber view at approximately 90°, the transesophageal, two-chamber view (at 60–90°) or the transesophageal long-axis view (at 120–150°).[1]

- Is there evidence of *severe* regurgitation, in particular systolic pulmonary venous flow reversal, large proximal jet width, and/or a large proximal convergence zone?

If transthoracic echo is inconclusive in answering these questions with confidence, TEE should be performed without delay. If an acute mitral prosthesis dysfunction is suspected, TEE should be used right away. The most important views are the lower transesophageal views,[1] which allow one to scan systematically the whole prosthesis and its circumference for abnormalities.

Doppler echocardiographic features of acute severe mitral regurgitation

Important Doppler features of *severe* mitral regurgitation to look for typically include the following:

- A regurgitation jet of at least moderate size by color Doppler. Eccentric jets represent more severe regurgitation than they appear to do. Because the size of the color Doppler jet depends directly on the pressure gradient, the jet actually decreases in size with diminishing systolic blood pressure and rising left atrial pressure, while the regurgitant lesion remains the same. As in chronic conditions, the jet size alone therefore may be misleading.
- Reversal or severe blunting of systolic pulmonary venous inflow. Pulmonary vein flow can be recorded by pulsed Doppler in the right upper pulmonary vein from the apical, four-chamber view or, by TEE, in the left or right upper pulmonary vein. Reverse flow in the pulmonary vein can often already be seen by color Doppler. This is a highly specific, but not very sensitive sign of severe regurgitation.
- A reproducible, large (over 1 cm^2) proximal acceleration zone (‘PISA’) on the ventricular side of the regurgitant lesion is usually seen even with the highest color Doppler aliasing velocities. For details on how to use quantitatively the proximal convergence zone to calculate the regurgitant flow rate or regurgitant orifice (which usually is neither practical nor necessary in the emergency setting), the reader is referred to systematic overviews.[5]
- If there is a reproducible, clearly delineated jet, its minimal width at its origin (‘proximal width’, ‘Vena contracta’) exceeds 7 mm. In many cases, however, as in prosthetic valve dehiscence, this width is not clearly visualizable. However, it is usually obvious that the jet width is large.
- A rare sign of very severe mitral regurgitation is a ‘shoulder’ in the continuous wave profile of mitral regurgitation, caused by a rapid decrease in late systolic velocities. Hence, the usually symmetric bell shape of the regurgitation profile becomes distorted, and looks more triangular. This notch reflects the massive left atrial pressure increase

in late systole due to the regurgitation, and therefore a late systolic decrease in ventriculoatrial pressure gradient.

Additionally, in acute mitral regurgitation, there is almost always morphologic evidence of severe disease. Specific features are discussed below. Importantly, the following typical features of severe *chronic* mitral regurgitation are missing in acute regurgitation:

- Regardless of the severity of regurgitation, neither the left atrium nor the left ventricle is necessarily enlarged. At least initially, sinus rhythm is mostly preserved. However, the presence of enlargement does not exclude acute regurgitation, since concomitant or previous disease (as in valve replacement) may have led to chamber enlargement.
- Global left ventricular dysfunction is not a typical feature of acute mitral regurgitation, and typically there is left ventricular hyperkinesis as a response to the volume load of acute regurgitation. However, left ventricular dysfunction does not exclude this condition, since there may be concomitant myocardial disease.

Specific causes of acute mitral regurgitation and their echocardiographic correlates

Infective endocarditis with valve destruction

Infective endocarditis can lead to perforation of a leaflet or to chordal rupture with flail of the affected leaflet. In mitral valve prostheses, it can lead to perforation or flail of bioprosthetic leaflets or to dehiscence of all types of prosthetic valves (see below). Usually, there are other morphologic signs of endocarditis: leaflet thickening, vegetations, pseudoaneurysms, or abscess. Although severe regurgitation is generally considered a relatively late event in endocarditis, staphylococci in particular may produce severe regurgitant lesions within a few days or less.

Papillary muscle rupture

This condition occurs in approximately 1% of acute infarcts, mostly with infarctions of the posterior circulation, in the first days after infarction, although it may occur very early. The posteromedial papillary muscle, which is variably perfused by the right or circumflex coronary artery,[2,3] is far more frequently affected than the anterolateral. Actual rupture of the whole papillary muscle

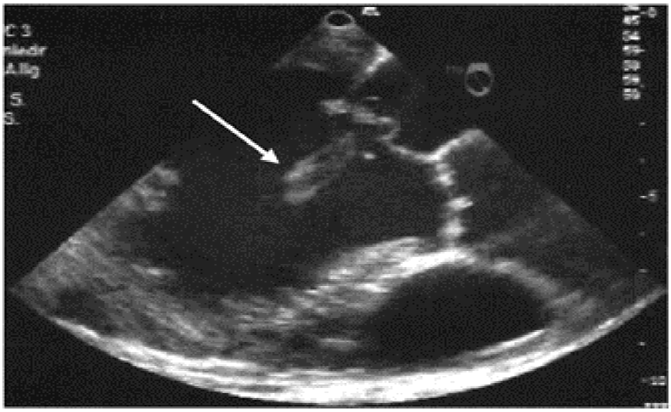


Figure 13.1 Papillary muscle rupture of a head of the posteromedial papillary muscle (arrow) following inferior myocardial infarction. Transgastric, two-chamber view at 110°.

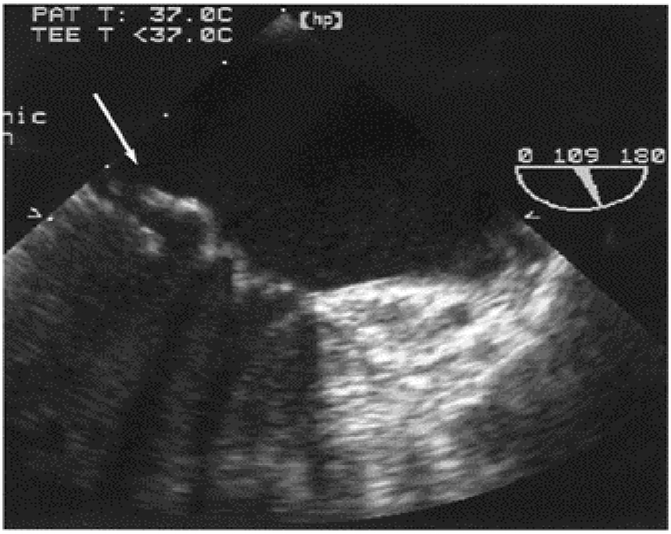


Figure 13.2 Severe mitral regurgitation due to degenerative chordal rupture and flail of the anterior leaflet (arrow) at 109°. The view

roughly corresponds to a transesophageal, two-chamber view.

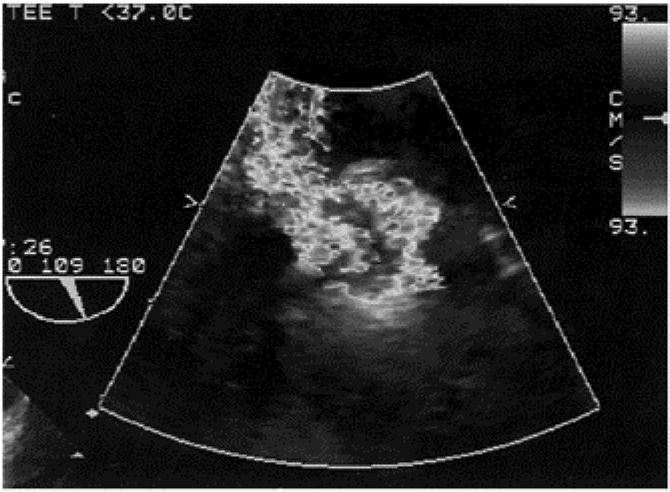


Figure 13.3 Color Doppler zoom image corresponding to Figure 13.2 shows a large proximal convergence zone, even with high aliasing velocity (93 cm/s), confirming severe regurgitation, as already predicted by the 2-D image.

is very rare and mostly catastrophic; what usually is designated papillary muscle rupture instead is the ischemic rupture of one of the about six heads of the posteromedial papillary muscle. It can be seen rapidly moving with the mitral leaflets, being displaced into the left atrium in systole and returning to the left ventricle in diastole (Figure 13.1). Transthoracic imaging usually is sufficient for diagnosis if there is a reasonable echo window. The size of the infarct-induced wall motion abnormality often is not very large, leaving global ventricular function

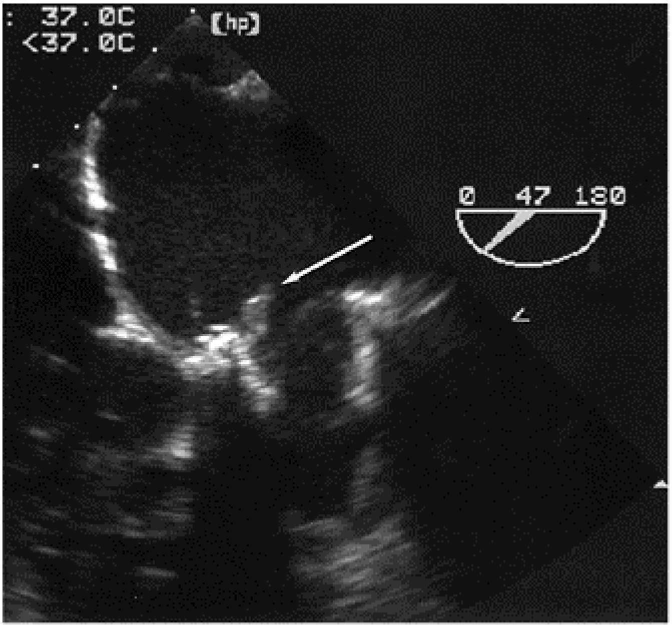


Figure 13.4 Degenerative leaflet flail (arrow) of a Carpentier-Edwards mitral bioprosthesis.

normal or near normal, and rupture typically occurs during the first days after onset of infarction, with rapid hemodynamic deterioration of the patient.

Degenerative chord a I rupture

Chordal rupture may occur as a degenerative complication or as a complication of infective endocarditis in mitral valve prolapse or, more rarely, in a normal valve. The uniform consequence is (partial or total) flail of the affected leaflet, with a regurgitant jet directed away from the affected leaflet (Figures 13.2 and 13.3). The ruptured chorda is often seen moving erratically in the left atrium during systole and in the left ventricle in diastole. The distinction between a degenerative and an endocarditic etiology of a ruptured chorda, especially in the presence of a diffusely thickened valve, as in classic mitral valve prolapse, usually cannot be made with confidence on echocardiographic grounds alone, unless clear vegetations or a perforation is present. The presence or absence of general signs of infective endocarditis (fever, positive blood cultures, and serum markers of systemic inflammation, such as elevated blood sedimentation rate or C-reactive protein) is of critical importance in these cases.

Mitral prosthetic dysfunction

With ever increasing numbers of patients with mitral valve replacement, this scenario is becoming increasingly important. Depending on the type of prosthesis, the following possibilities for acute mitral prosthetic regurgitation exist:

• Bioprosthetic degeneration or endocarditis. The time-dependent wear-and-tear lesions of bioprostheses may often remain entirely silent before a large tear suddenly manifests itself as torrential regurgitation. The degenerative tear can lead to leaflet prolapse or flail, or to perforation. In contrast, infective endocarditis affects bioprosthetic leaflets in a similar manner to native valve leaflets, leading to thickening, vegetations, or perforation. The differential diagnosis of pure degeneration and endocarditic lesions is not possible by echocardiography alone; degenerative and endocarditic lesions of a bioprosthesis often are indistinguishable. For example, tears can lead to prolapsing leaflet segments (Figure 13.4) that may mimic vegetations, and endocarditic leaflet thickening may closely resemble degenerative changes. As pointed out above, the presence or absence of general signs of endocarditis is important to ascertain the etiology. Importantly,

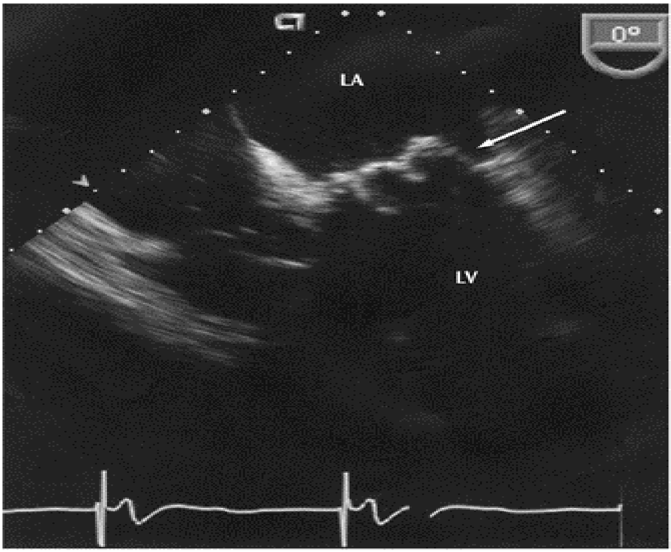


Figure 13.5 Lateral dehiscence (arrow) of a mitral bioprosthesis. Transesophageal, four-chamber view in systole, showing displacement and tilting of the prosthesis toward the left atrium. (Reproduced with permission

from Lambertz H, Lethen H, *Atlas der transösophagealen Echokardiographie,* Stuttgart: Thieme, 2000.) LA: left atrium; LV: left ventricle.

the size of the left atrium and ventricle, as well as the level of pulmonary hypertension, is influenced by pre-existing disease and therefore must be interpreted with caution with respect to the severity of acute mitral regurgitation.

- Prosthetic ring endocarditis with prosthetic dehiscence. Infective endocarditis of valvular prostheses preferentially leads to ring abscesses, particularly, but not exclusively, in mechanical prostheses. Ring abscesses destroy the anchoring of the prosthesis in its bed. Depending on the extensiveness of the abscess, regurgitation may ensue, ranging from paravalvular leakage to dehiscence, defined as abnormal mobility ('rocking') of the whole prosthesis, to embolism of the entire prosthesis. Abnormal mobility can be best seen by careful back-and-forth review of the prosthesis motion on 2-D echo over a full cardiac cycle. By TEE, usually the dehiscence can be directly identified as a break in continuity between the prosthethic ring and the paravalvular tissue, giving rise to a large paraprosthetic regurgitant jet.
- Dehiscence may also occur as the result of suture insufficiency in the early postoperative period (Figure 13.5).
- Mechanical (and rarely, biologic) prosthetic thrombosis often leaves the occluder or leaflets in a fixed, half-shut position, leading to both severe stenosis and regurgitation. By careful apical imaging, abnormal occluder mobility can often be ascertained in tilting-disk or bileaflet prostheses in the mitral position. In case of doubt, TEE should be performed. Unless cardiac output is already very low, continuous-wave spectral Doppler shows clearly elevated diastolic transprosthetic velocities yielding gradients well above 5±3 mmHg, which is the normal range for mechanical prostheses in the mitral position at normal heart rates. Of course this is partly due to the additional regurgitant volume flowing through the prosthesis in diastole. In many cases, thrombus can be directly seen on the atrial side of the prosthesis by TEE, but lack of clearly visualizable thrombotic material by no means excludes thrombosis of the valve! The decisive finding of prosthetic thrombosis is impaired leaflet mobility, not thrombus visualization. The differential diagnosis of pannus interfering with the occluding mechanism is almost impossible to exclude by echo alone, although it has been reported that pannus is more echodense than thrombus,[4] and the diagnosis should rather be made by considering the acuity of the condition, anticoagulation status, embolic events, etc.
- A very rare cause of acute massive prosthetic dysfunction is prosthetic strut fracture leading to embolization of the occluder. This has been seen in a series of Björk-Shiley tilting disk valves (60–70° convexoconcave mitral prosthesis no. 29 implanted during the years 1979–86) and exceptionally in other valves. The occluder often embolizes to the abdominal aorta. The event is mostly fatal due to overwhelming mitral regurgitation.

Differential diagnosis

The combination of a well-pumping left ventricle with dyspnea and/or pulmonary edema should bring to mind the following important differential diagnoses apart from acute mitral regurgitation:

- hypertensive crisis
- atrial fibrillation with rapid ventricular rate
- cardiac tamponade
- acute aortic regurgitation
- constrictive pericarditis
- acquired shunt lesion, such as post-infarction ventricular septal defect
- noncardiac pulmonary edema (as by fluid volume overload) and others.

While mild mitral regurgitation is frequent even in normals, and the regurgitant jet of mild or moderate mitral regurgitation by color Doppler may look relatively impressive with systemic hypertension, the absence of a structural abnormality of the mitral valve largely excludes acute severe mitral regurgitation.

References

1. Flachskampf FA, Decoodt P, Fraser AG, Daniel WG, Roelandt JRTC. Recommendations for performing transesophageal echocardiography. Eur J Echocardiogr 2001; 2;8–21.
2. Estes EH, Dalton FM, Entman ML. The anatomy and blood supply of the papillary muscles of the left ventricle. Am Heart J 1966; 771:356–62.
3. Voci P, Bilotta F, Caretta Q, Mercanti C, Marino B. Papillary muscle perfusion pattern: a hypothesis for ischemic papillary muscle dysfunction. Circulation 1995; 91:1714–18.
4. Barbetseas J, Nagueh SF, Pitsavos C, Toutouzas PK, Quinones MA, Zoghbi WA. Differentiating thrombus from pannus formation in obstructed mechanical prosthetic valves: an evaluation of clinical, transthoracic and transesophageal echocardiographic parameters. J Am Coll Cardiol 1998; 32:1410–17.
5. Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. J Am Soc Echocardiogr 2003; 16:777–802.

14
Echocardiography in acute myocardial infarction

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Key points

- Echocardiography should be used in all patients with acute myocardial infarction for diagnosis, functional assessment, detection of complications, and/or prognosis.
- Echocardiography may miss small, nontransmural infarctions.
- Regional dyssynergy associated with preserved wall thickness in diastole may be due to ischemia and acute myocarditis, not only infarction.
- The value of echocardiography as a diagnostic tool is highest in patients with suspected acute myocardial infarction with atypical clinical presentation and/or nondiagnostic electrocardiogram.
- Measuring ejection fraction in the acute phase of the infarction may not reflecdt true functional loss, although a low postinfarction ejection fraction is a strong predictor of poor outcome.
- Dysynergic myocardial regions may be viable and may have the potential of functional recovery. Viability can be assessed early by echocardiography-based techniques.
- Initial left ventricular volumes, wall motion score index, infarct zone viability, and diastolic filling assessed by deceleration time carry important prognostic information.

In patients with suspected or evolving acute myocardial infarction, echocardiography facilitates diagnosis and differential diagnosis, detects complications (Chapter 15) and provides valuable information regarding infarctrelated artery, functional infarct size, prognosis, and the effects of therapy (Table 14.1).

Table 14.1 The role of echocardiography in acute myocardial infarction

1.	Diagnosis
2.	Differential diagnosis
3.	Functional infarct size
4.	Infarct-related artery
5.	Left ventricular function
6.	Viability
7.	Prognosis
8.	Effects of therapy

As a handy, widely available, and cost-effective imaging technique, echocardiography should be used in almost all patients with acute myocardial infarction for at least one of the reasons listed above. The collected information, whether assessed quantitatively or qualitatively, is of incomparable value for accurate estimation of risk and for guiding management. Even in patients traditionally considered as low-risk, echocardiography may reveal unexpected left ventricular dysfunction due to an extensive dysynergic zone.[1] However, it should be emphasized that echocardiographic examination in unstable patients with ongoing chest pain in the emergency setting is a highly demanding procedure that requires both excellent technical skills to obtain adequate images in a stressful environment and the ability to interpret findings quickly and accurately.

Two technological developments allow improved endocardial definition by two-dimensional echocardiography, which is crucial for accurate regional wall motion analysis: second harmonic imaging, and intravenous echocardiographic contrast agents. These techniques allow fair endocardial delineation and left ventricular opacification in the majority of difficult cases with poor echocardiographic images.[2,3] However, these tools should not be considered as substitutes for excellence in regional wall motion assessment. Experienced supervisors are able to provide training that may result in dramatic improvement in regional wall motion abnormalities readings in a short period of time.[4] In addition, intracoronary[5] or intravenous[6] myocardial contrast echocardiography (MCE) provides important data regarding the area at risk after coronary occlusion, regional coronary flow reserve, myocardial viability, and the functional outcome.

Although in almost all cases standard two-dimensional and Doppler transthoracic examination provide information that may be effectively used for decision making, transesophageal echocardiography (TEE) may also be helpful, especially in critically ill patients. In experienced hands, TEE is safe and feasible. It may be of value particularly in differential diagnosis, to exclude other life-threatening conditions with similar clinical presentation, such as aortic dissection (Chapter 12) or massive pulmonary embolism (Chapter 8).

Since the clinical course of acute myocardial infarction is highly variable, serial echocardiographic studies may be needed to assess actual risks and to correct therapeutic strategy throughout the acute phase. In addition, followup studies provide unique insight into the natural history of left ventricular systolic and diastolic function and the effects of reperfusion therapy.

Echocardiographic signs of myocardial infarction

Myocardial ischemia or infarction causes abnormalities in regional wall motion of the left ventricle[7] that can be easily documented by echocardiography. The key echocardiographic sign of acute myocardial ischemia/necrosis is regional dyssynergy associated with preserved wall thickness in diastole.[8–10] Over time, necrotic myocardial segments typically become thin and highly reflective. In contrast to infarction, ischemia, by definition, causes transient wall motion abnormalities.

In patients with ongoing myocardial infarction, two-dimensional echocardiography may reveal different degrees of dyssynergy: hypokinesis, akinesis, or dyskinesis. The degree and the magnitude of dyssynergy caused by actual infarction are related to the

location of the culprit lesion in the infarct-related artery, the presence or absence of collateral circulation, and the extent of coronary artery disease.

It has been shown that dyssynergy detectable by echocardiography occurs if resting coronary flow is reduced by >50%,[11–13] if >20% of myocardial thickness is jeopardized by actual ischemia/necrosis,[14,15] or if at least 1–6% of the left ventricular mass is involved.[16] Thus, echocardiography may miss myocardial infarction if it is very small and/or limited to the thin endocardial layer (small nontransmural infarctions).[9,10,16]

Echocardiographic diagnosis of acute myocardial infarction

In patients with suspected acute myocardial infarction, the accuracy of diagnostic information obtained by echocardiography is related to local expertise, clinical circumstances, and the specific patient population evaluated. It is well known that the sensitivity, specificity, and accuracy of any diagnostic technique are strongly influenced by the pretest likelihood of the disease (Bayesian theory). Therefore, different results can be expected in the coronary care unit patient population from patients examined in the emergency department, who represent a much less selected group of individuals. Successful technique, however, should identify low-risk patients without compromising the detection of those at high risk of coronary events and complications.[17]

As pointed out before, in acute myocardial ischemia/ necrosis, echocardiography reveals regional dyssynergy associated with preserved wall thickness in diastole and normal myocardial reflectivity. A thin, dysynergic, and bright or highly reflective myocardial wall strongly suggests scar due to old infarction. However, in patients with acute chest pain, it is often impossible to distinguish regional dyssynergy caused by ischemia, necrosis or myocardial inflammation, on the basis of two-dimensional echocardiogram alone.[10,18–22] Review of the studies of echocardiography in diagnosis of myocardial infarction reveals that it seems to be sensitive in the diagnosis of acute infarction or ischemia during anginal pain.[10,19–23] The incremental value of echocardiography as a diagnostic tool is highest in patients with suspected acute myocardial infarction presenting with atypical clinical presentation and/or a nondiagnostic electrocardiogram.[24]

Overall, sensitivity of the technique is high, over 90%, but specificity and positive predictive value are more variable and less convincing,[23,24] indicating interpretation difficulties in the emergency setting. As a consequence, confusion and frequent overdiagnosis may occur in attempts not to miss those with true infarction. On the other hand, the absence of segmental wall motion abnormalities in patients without history of infarction or known coronary artery disease has negative predictive value as high as 99%.[18,19] Although it

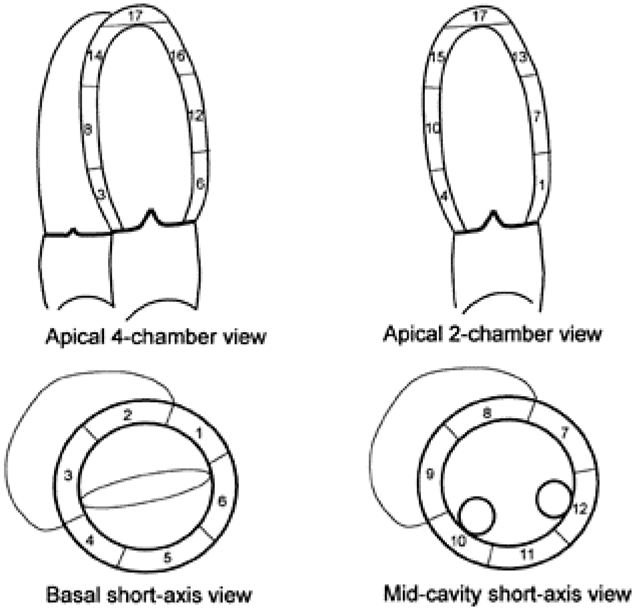


Figure 14.1 Coronary artery territories. Left anterior descending coronary artery supplies anteroseptal and apical septa I segments (2, 8, and 14), anterior free wall (1 and 7), anterior apical segment (13), and true apex (17). Right coronary artery supplies inferior free wall (4 and 10), inferior septum (3 and 9), and inferior apical segment (15). Left circumflex coronary artery supplies inferolateral (5 and 11), anterolateral (6 and 12), and lateral apical segment (16). There is overlap between right coronary artery and left circumflex coronary artery territories, according to the coronary artery dominance.

should be expected that a few small subendocardial injuries will be missed using echocardiography alone, importantly, the prognosis in patients with acute coronary syndromes and normal echocardiogram is favorable.

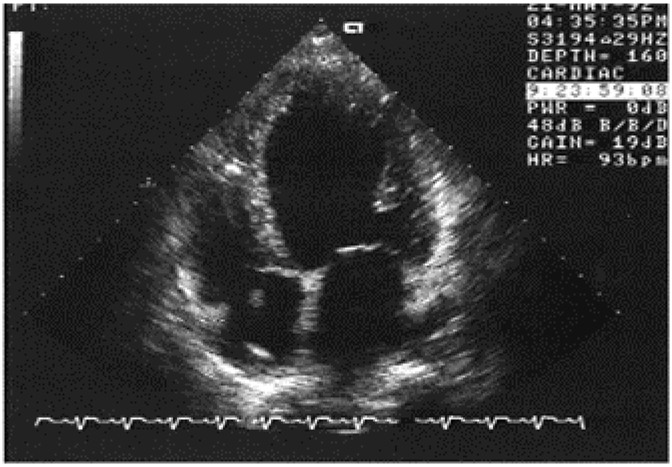
Differential diagnosis

Echocardiography provides critical information for differentiation of other conditions that may be associated with acute chest pain and/or hemodynamic instability where recommended therapeutic strategies are strikingly different and where misdiagnosis and mistreatment may be life-threatening (that is, acute aortic syndrome versus acute ST-elevation myocardial infarction). The situation may become even more confusing when these conditions occur simultaneously, as in the case of acute coronary artery obstruction due to dissection of vessel wall or obstruction with intimal membrane in acute type I aortic dissection.

Apart from acute infarction, regional dyssynergy caused by coronary artery disease can be detected during transient anginal attack, in chronic ischemia (hibernated myocardium) or in patients with old infarctions (myocardial scar). In addition, acute myocarditis, cardiomyopathy, and left bundle branch block may be associated with regional wall motion abnormalities indistinguishable from those that occur due to obstructive coronary artery disease. Of note, contrary to common opinion, a substantial proportion of patients with clinically significant acute myocarditis reveal segmental, rather than diffuse, wall motion abnormalities, quite similar to typical echocardiographic presentation of an acute coronary event.[25–27] However, aneurysms are not seen in myocarditis (except in rare cases of Chagas's myocarditis).

Infarct-related artery

Actual distribution of dysynergic segments on the two-dimensional echocardiogram reflects the location of the culprit coronary artery lesion (Figure 14.1).[10,21,28–33] if the left anterior descending coronary artery is involved, dyssynergy of the anterior wall, anterior septum, and left ventricular apex is present. The extent of dyssynergy is determined by the level of the infarct-related artery obstruction: the more proximal the lesion, the more extensive are wall motion abnormalities. Lateral wall dyssynergy points to the left circumflex artery as an infarct-related artery, while dyssynergy of the inferior wall and inferior septum suggests right coronary artery lesion.[33] Of note, there is substantial overlap in segmental distribution of the territories supplied by the right coronary artery and left circumflex coronary



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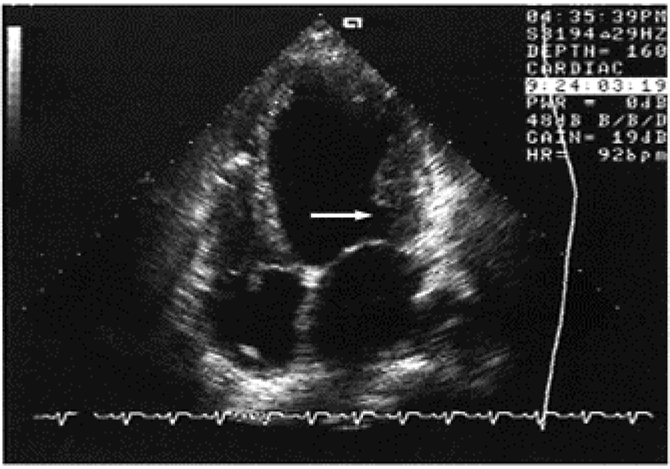

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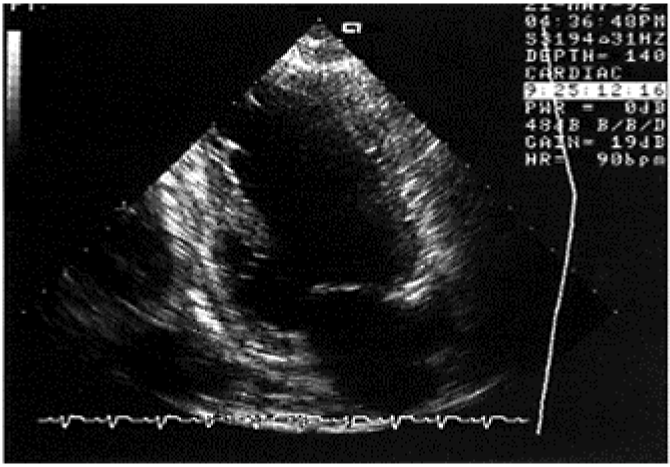
Figure 14.2 Functional infarct size. Example of a patient in cardiogenic shock from extensive anterior myocardial infarction. Note that only the basal parts of the anterior and lateral wall are moving in systole (marked with arrows). Panels A (diastole) and B (systole) show apical four-chamber view; C (diastole) and D

(systole) show apical two-chamber view.

artery, depending on the coronary artery dominance in a particular patient. Remote dyssynergy detected in segments out of a single coronary artery perfusion zone typically indicates mutivessel coronary disease.[33]

Functional infarction size

The extent of dysynergic myocardium in evolving myocardial infarction determines its functional size and has a direct impact on the degree of hemodynamic compromise and the prognosis (Figure 14.2). The extent of dyssynergy correlates well with the anatomical size of the infarction or jeopardized myocardium.[34–37] However, information obtained by segmental wall motion analyses tends to overestimate true anatomical infarction size.[34–36, 38] The nonfunctional zone consists of the actual necrosis zone, segments jeopardized by actual ischemia, stunned and hibernated segments, and dysfunction from previous infarctions. Thus, functional infarct size is often considerably larger than the anatomical size of actual necro-



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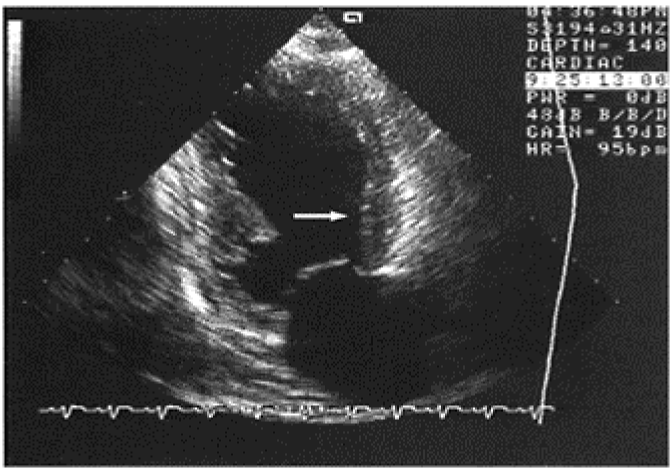

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sis.[38–41] Frequently, hypercontractility of the segments not affected by actual infarction can be noted (see below); as a consequence, the global ejection fraction can be maintained in the normal range, or it is only slightly reduced during the acute phase. In patients with complicated clinical course, repeated echocardiographic examinations in the coronary care unit may replace hemodynamic monitoring in a majority of cases, since they provide useful information on changes in left ventricular function and may detect complications to assist decision making regarding therapeutic intervention.

Of note, many patients without clinical evidence of hemodynamic instability may have significant wall motion abnormalities,[1] indicating a risk of early left ventricular remodeling and an associated poorer prognosis. These patients identified by serial echocardiographic examinations require an aggressive management strategy.

Finally, it is important to remember that there is frequent discordance between severity of electrocardiographic changes and true extent of myocardial damage. The

electrocardiogram may over- or underestimate the amount of dysfunctional myocardium in both the acute and chronic phases.

Evaluation of left ventricular function

Functional impairment of the left ventricle in acute myocardial infarction may be assessed in different ways. Changes in different parameters of left ventricular systolic and diastolic function are important features of acute myocardial infarction. Serial echocardiographic assessment is necessary for early detection of deterioration or to document improvement of these parameters over the time of and/or after therapeutic interventions. In addition, the absolute value of these parameters as well as the pattern of their changes over time carry major prognostic information in postinfarction survivors (see below).

The improvement of segmental function over time is a well-known phenomenon after myocardial infarction, especially in the reperfusion era. This improvement has been shown to be related mainly to timely administered reperfusion therapy, percutaneous coronary intervention, infarct-related artery patency, effective tissue reperfusion, and viability of the infarcted segments. These factors are obviously interrelated. They probably, in different ways, reflect the same basic principle, that is, preserved myocardial viability and halted necrosis process.

Systolic function

Global left ventricular function, assessed by ejection fraction, may be preserved even in large infarctions during the acute phase as a consequence of the compensatory hypercontractility of noninfarcted segments. Hyperkinesia of the remote areas has been reported in 4.5–67% of patients after acute myocardial infarction, the incidence being related to the time when the examination was performed (it declines with time after infarction) and the extent of the coronary artery disease (lower incidence in multivessel disease).[42–46] Hyperkinesia may disappear during the first days following an infarction, especially after successful revascularization in patients with single-vessel disease.[46–50] In some studies, hyperkinesia was observed more often in patients with anterior infarction,[44,51] probably due to easier visual detection of hyper-kinesia in the inferior wall. In addition, the presence of bundle branch block makes the detection of hyperkinesia more difficult. Moreover, it can be seen more often in patients treated with thrombolytic therapy,[51] correlating with increased TIMI (thrombolysis in myocardial infarction) grade flow in the infarct-related artery.[52] Therefore, measuring the ejection fraction in the acute phase may not reflect true functional loss, although low postinfarction ejection fraction is a strong predictor of poor outcome.[53–55]

Infarction size is a major determinant of left ventricular ejection fraction. Since anterior infarctions tend to be larger and are more frequently coupled with infarct expansion, they often cause greater reduction in global left ventricular ejection fraction.[56,57] It has been recently demonstrated that for infarctions of similar size, apical involvement is associated with lower left ventricular ejection fraction.[58] These findings

indicate that the infarction site, independently of its size, may have an important impact on left ventricular systolic function.

A high wall motion score index, which reflects extensive and severe actual dyssynergy, is generally associated with deprived global left ventricular function, and it indicates a potentially complicated course and poor prognosis.[10,18,22,28,59–62]

Increased initial left ventricular end-diastolic and end-systolic volumes indicate extensive myocardial damage[63] and require serial echocardiographic evaluation for early detection and estimation of the presence and the degree of adverse left ventricular remodeling.[64–66]

Failure to achieve adequate microvascular reperfusion at tissue level (the ‘no-reflow’ phenomenon) as detected by MCE during acute myocardial infarction[67,68] is consistently associated with impaired functional recovery of affected myocardial segments and a worse clinical outcome.[67–73]

Contractile recovery occurs earliest in well-reperfused segments, involving 40% of these segments before the end of week 2.[74,75] However, significant recovery can still be observed at week 6 in segments with homogeneous contrast enhancement, indicating resolved myocardial stunning.[67,76] Importantly, up to 25% of segments with heterogeneous contrast enhancement also reveal wall motion recovery within the first 6 weeks, while the absence of tissue perfusion is highly predictive of the permanent contractile impairment of infracted segments.[75]

Diastolic function

A few important caveats should be noted in the assessment of left ventricular diastolic function by Doppler echocardiography in the setting of acute myocardial infarction. Left ventricular filling is affected by numerous factors that may have influence on left atrial to left ventricular pressure gradient in diastole. Therefore, it is very difficult to assess diastolic function in individual patients from ventricular filling only, because it may appear similar in different clinical settings that change the pressure gradient in the same way.

Almost all Doppler indices of left ventricular diastolic function are affected by a number of physiologic factors,[77,78] such as heart rate,[79] that can change ventricular filling regardless of diastolic function. In addition, these indices are dependent on left ventricular systolic

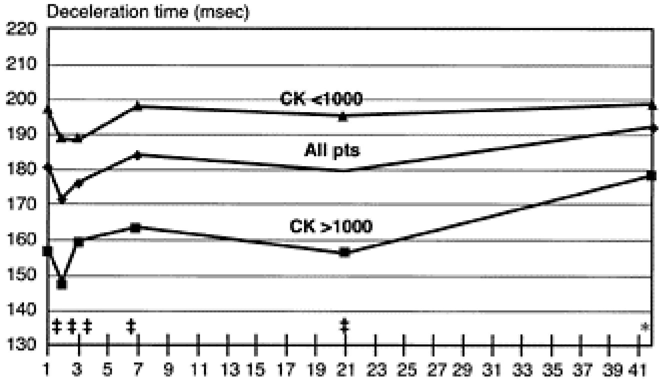


Figure 14.3 Doppler-derived deceleration time decreases in first 24–48 h after acute myocardial infarction, but returns to normal within several days, indicating biphasic changes in left ventricular chamber stiffness. Deceleration time is shorter in patients with large (CK >1000 i.u.) than small infarcts. CK: creatine kinase. (Reproduced from Popović AD et al. Am J Cardiol 1996; 77:361–4[85] with permission from Excerpta Medica, Inc.)

function.[79,80] Finally, therapy may produce major changes of indices of diastolic function simply by varying loading conditions.[81,82]

However, it appears that the early filling deceleration time is less dependent on heart rate, contractility, and afterload[83] and reliably reflects left ventricular chamber stiffness.[84]

In patients with acute myocardial infarction, biphasic changes in left ventricular chamber stiffness have been demonstrated by Doppler echocardiography.[85] Increased left ventricular chamber stiffness, denoted by decreased early filling deceleration time, can be detected 24–48 h after myocardial infarction, but it returns to normal within several days.[85] The increase of chamber stiffness is higher in large/anterior infarcts, and appears to be independent of left ventricular systolic function (Figure 14.3).[85] Importantly, it has been shown that a normal ratio of early to atrial filling (E/A) can be found in half of the patients after myocardial infarction.[85,86] A very few patients (with the largest infarcts) have a marked increase of early to atrial filling ratio of >2, indicating significantly increased left ventricular filling pressure.[85] Thus, the appearance of early to atrial filling ratio after myocardial infarction may be misleading in differentiating patients with normal and impaired left ventricular diastolic function.

Viability

As a consequence of myocardial infarction, alterations in myocardial tissue structure and composition occur, leading to changes in the acoustic properties of affected myocardial segments. With a standard, resting, two-dimensional echocardiographic examination, however, early differentiation of nonfunctional infarcted myocardium with occluded infarct-related artery from reperfused, stunned, or hybernating myocardium with potential for functional recovery is not possible. The principal therapeutic aim in acute myocardial infarction is to preserve as much jeopardized myocardium as possible. To determine further management strategy after infarction, information about infarcted tissue properties, that is, whether infarcted segments are still viable or not, can be collected early. These data are as important as information on vessel patency, since the patent infarctrelated artery, on coronary angiogram, does not guarantee the viability of perfused segments.

In fact, viable myocardial tissue is almost always present in segments affected by actual infarction. Echocardiographic modalities for detection of viability include low-dose dobutamine and dipyridamole echocardiography, tissue characterization, and detecting of functional improvement of previously dysynergic segments on follow-up studies that occurs spontaneously or after revascularization.[87–90]

Viability can indicate the therapeutic success of acute intervention, and it has a beneficial impact on survival in those suitable for revascularization.[91]

Assessment of myocardial viability early after myocardial infarction by dobutamine echocardiography

It is now well established that in patients with coronary artery disease, impaired left ventricular function at rest is not necessarily an irreversible process, since dysynergic myocardial regions may be viable and may have the potential to recover function.[92,93] This can occur in different states: during a short period of reversible ischemia, in nontransmural infarction, and myocardial stunning and hibernation. Identification of these abnormal myocardial conditions is difficult and usually requires more than one technique. It has to be emphasized that the occurrence of pure transmural stunning or hibernation is infrequent; commonly, there is a mixing of subendocardial scar with a variable amount of viable subepicardium, which may be in one of the states listed above.

The identification of viable myocardium with dobutamine echocardiography is based on the principle that the presence of a contractile reserve of dysynergic segments may be evoked by inotropic stimulation. The bulk of data has demonstrated that contractile reserve by dobutamine is accurate in predicting functional recovery after infarction, even in patients who do not undergo revascularization.[69,94–103] It has been suggested, therefore, that the contractile reserve may be unmasked by low-dose dobutamine infusion in regions exhibiting stunned myocardium. Whether dobutamine echocardiography is similarly efficacious in the evaluation of the hibernating myocardium is uncertain, because adequate animal models of chronic hypoperfusion to test this hypothesis are lacking. As hibernating myocardium represents a delicate balance among flow, function, and viability, it is likely that the increased demands of inotropic stimulation in some patients will overwhelm the limited flow reserve and result in myocardial ischemia. This may

explain the common finding of a biphasic response to dobutamine, in which low doses elicit an increase in regional systolic wall thickening, but higher doses exhaust flow reserve and produce ischemia, resulting in a reduction in wall thickening.[104] Furthermore, in the natural history of hibernation, cellular dedifferentiation and slippage of myofibrillar units may result in significantly reduced or absent responsiveness to catecholamine stimulation.[105]

A more controversial concept is whether inotropic stimulation can cause ongoing augmentation of transmural systolic function after exhaustion of flow reserve and during active ischemia, particularly in the subendocardial region.[106]

Despite these conceptual limitations, a number of published reports suggest that dobutamine echocardiography is accurate in detecting myocardial viability also in postinfarction patients with critically stenosed, infarctrelated arteries.[94–100,102] In patients with acute myocardial infarction with either successful pharmacologic or mechanical reperfusion therapy, the response of post-ischemic myocardium to dobutamine is influenced by the extent of necrosis, the severity of residual stenosis, the size of the risk area, and the extent and magnitude of collateral blood flow.[107] In the absence of a residual stenosis, the transmural extent of necrosis determines the degree of systolic thickening for any given dose of dobutamine. The ideal situation is the presence of a small area of necrosis with the coexistence of stunned myocardium. In such patients, nuclear imaging will indicate normal or normalized perfusion, and dobutamine will increase wall thickening even at low doses. In the presence of more extensive necrosis, systolic thickening starts increasing only at moderate doses, with further increases at high doses.

On the other hand, if the necrosis is transmural, wall thickening does not increase at any dose. Thus, in the absence of a significant residual stenosis, as in a patient successfully treated by primary coronary angioplasty, no response at any dose of dobutamine suggests nonviable myocardium.[107] Experimental studies have demonstrated that there is an excellent relationship between the transmural extent of necrosis and percent wall thickening during low-to-moderate doses of dobutamine infusion in the setting of myocardial necrosis coexisting with post-ischemic myocardial dysfunction and no-flow-limiting residual stenosis: the higher the systolic thickening, the smaller is the infarct size.[108]

These results have been recently confirmed in a clinical model of reperfused acute myocardial infarction.[103] In patients successfully treated with primary coronary angioplasty and no-flow limiting residual stenosis in the infarctrelated artery, low-dose dobutamine echocardiography (up to 10 µg/kg per min) performed early (3 days) after the index infarction was able to identify the presence and the extent of viable myocardium. The extent of contractile reserve, expressed as infarct zone wall motion score index changes at peak dobutamine, correlating well with the spontaneous recovery at late follow-up.

Furthermore, dobutamine echocardiography was able to predict the quantitative improvement in global systolic ventricular function, which was related to the number of myocardial segments with contractile reserve and to the magnitude of their functional improvement.[103] This relationship may be compromised if the infarct-related artery is severely stenotic. In this case, jeopardized myocardium can be identified by a biphasic response (improvement of contractility at low dose and worsening at high dose) or by a new dyssynergy in adjacent segments within the infarct-related area. In the presence of a

flow-limiting stenosis, ischemia may occur, and wall thickening diminishes despite the presence of viable myocardium. Therefore, the induction of ischemia in a dysfunctional segment suggests the presence of both viable myocardium and significant residual stenosis, even in the absence of a biphasic response. For the same reasons, it must be recognized that persistence of akinesis in the affected region during the dobutamine test does not necessarily imply absence of residual viability. Finally, ischemia at a distance from the affected area, detected by dyssynergy in response to high-dose dobutamine, correlates with the presence of multivessel coronary artery disease.

Clinically relevant myocardial viability

Recently, contractile reserve during dobutamine echocardiography has been increasingly used for predicting postischemic reversible dysfunction[94–104] and has been proposed as a simpler alternative to the more sophisticated and expensive nuclear technique.[109,110] The response of viable but stunned myocardium, even in the early convalescent phase after acute myocardial infarction, has been shown to correlate with functional recovery of dysfunctional myocardial segments. Recent data obtained in patients with acute myocardial infarction show that also hypoperfused viable myocardium supplied by a nearly occluded infarct-related artery can increase contractility in response to low-dose dobutamine infusion, even though the improvement was not consistently associated with an increase in myocardial blood flow.[102]

There is no information indicating the ‘minimum amount’ of viable myocardium needed to have an impact on prognosis, and this may vary by location. In a largescale, multicenter study of 778 survivors of an uncomplicated acute myocardial infarction and reasonably preserved resting baseline function, the presence of myocardial viability assessed by dobutamine echocardiography was associated with a higher risk of unstable angina, but not of nonfatal reinfarction and death.[111] In contrast, another recent prospective study[112] has shown a protective effect of myocardial viability detected with dobutamine echocardiography: the absence of contractile reserve by dobutamine was a strong independent predictor of a poor outcome. These apparently different results may be explained by different subsets of patients enrolled in each study. In the presence of a small-to-moderate necrosis with coexistence of stunned myocardium and no flow-limiting coronary stenosis, functional recovery is the rule and myocardial viability plays a protective role in clinical outcome. This hypothesis is supported by the finding that patients with extensive dyssynergy and myocardial viability have a prognosis similar to patients with small infarcts.[112] Other patients probably have hibernating myocardium superimposed on myocardial stunning due to the presence of significant residual stenosis or multivessel disease. In this situation, myocardial viability may disappear and extensive necrosis may occur, especially if early revascularization is not performed or is not followed by microvascular preservation.

Finally, the relation between myocardial viability and clinical outcome may be more complex than usually believed. It is generally postulated that only recovery in resting regional function denotes clinically relevant viability. However, although recovery in resting function is the best clinical outcome, there may be other advantages of having nonischemic viable myocardium. The presence of viable myocardium in the outer layers of the ventricular wall may in fact contribute to the maintenance of left ventricular shape

and size by preventing infarct expansion and subsequent heart failure, and thus reducing late ventricular arrhythmias and mortality after myocardial infarction. Recent data[113,114] show that the degree of left ventricular dilation after reperfused acute myocardial infarction is related to the extent of residual myocardial viability in the infarct zone assessed by low-dose dobutamine echocardiography. The presence of a relatively large amount of viable myocardium in the infarct zone, therefore, strongly contributes to the maintenance of left ventricular shape and size by preventing infarct expansion, and these findings appear to be independent of the infarct size and patency of the infarct-related artery.[113] These observations expand the role of the clinical and prognostic impact of myocardial viability in survivors of an acute myocardial infarction.

Information regarding microvascular integrity, obtained by MCE, and response to low-dose dobutamine infusion, assessed by echocardiography, may be used to predict irreversible damage and chances for functional recovery.[69] Patients with no myocardial contrast opacification revealed no response to dobutamine and no functional recovery, while various degrees of myocardial contrast opacification may be detected in both those with and without contractile improvement elicited by dobutamine (Chapter 10).[69]

Tissue characterization

In the image-processing field, a number of methods for texture characterization have been developed over the years and have been used in medicine for the assessment of different pathologic conditions of the myocardium. Recently, it has been shown that transient short-lasting myocardial ischemia is associated with an abrupt increase in myocardial reflectivity detectable by videodensitometric analysis applied to standard transthoracic echocardiographic images.[88] It has also been shown that the cyclic variation of relative integrated backscatter can be used to diagnose recent myocardial infarction.[89] Additionally, ultrasonic tissue characterization was successfully used for diagnosis of acute myocardial infarction in the coronary care unit, showing comparable results with standard two-dimensional echocardiography.[90]

Recently, cyclic, gray-level variation detectable in dysynergic segments on standard echocardiographic images was found to be a marker of myocardial viability.[115] By Wavelet image decomposition for myocardial tissue characterization,[87] no difference in myocardial texture exists between normal segments and reperfused

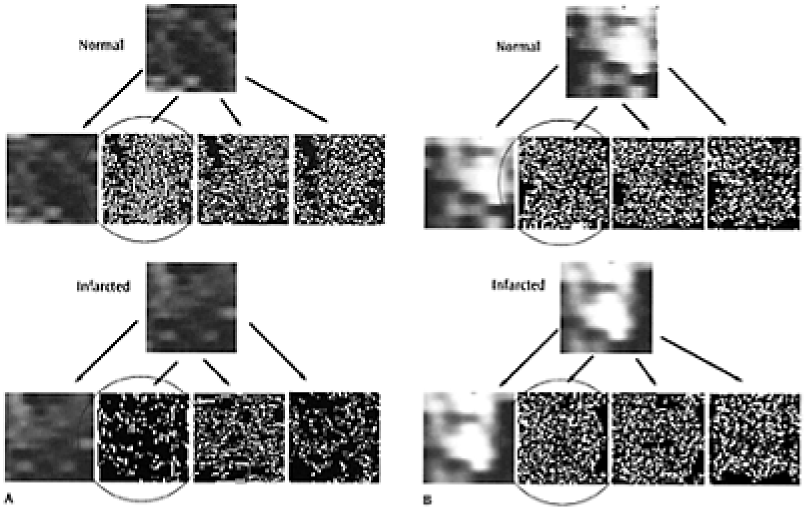


Figure 14.4 Tissue characterization by the Wavelet method. With nonreperfused myocardium (A), a clear difference between samples from normal and infarcted areas can be easily seen in the vertical-edge decomposed image (circled). However, no difference was noted between the normal and infarcted areas in reperfused myocardium (B). (Reproduced from Neskovic AN et al. Circulation 1998; 98:634–41[116] with permission from Lippincott Williams and Wilkins.)

infarcted segments, whereas a significant difference between normal myocardium and nonreperfused infarcted segments has been found as early as day 2 after infarction (Figure 14.4).[116] In addition, the majority of patients classified as reperfused by the Wavelet method showed regional functional improvement in follow-up studies, indicating that functional recovery of infarcted segments can be predicted by this method.[116] This method has the potential to detect viable myocardium early in the postinfarction period, providing additional information compared to serial observation of regional wall motion improvement over time.

Prognosis

It has been well known for years that a two-dimensional echocardiogram obtained immediately after admission to the coronary care unit[32] and/or before hospital discharge carries important prognostic information.[117] Numerous echocardiographic parameters have been used for risk stratification after acute myocardial infarction (Table 14.2).

Reduced left ventricular ejection fraction is associated with increased morbidity and mortality.[53–55,61] However, among patients with similar reduction of left ventricular ejection fraction after infarction, long-term prognosis is worse in those with higher left ventricular end-systolic volume.[118]

Large initial end-diastolic and end-systolic volumes are important predictors of left ventricular remodeling.[63,64, 66,119] Importantly, relatively small increases in left ventricular volumes are associated with five- to sixfold increase of the risk of cardiac death.[63] Quantitative measurements of left ventricular volume may predict complications after acute myocardial infarction. An end-systolic volume index of >35 ml/m^2 is associated with left ventricular thrombus formation during the acute phase.[120]

Infarct expansion, that can be easily detected by serial echocardiographic studies during the first days after acute

Table 14.2 Echocardiographic parameters that may be used for the assessment of prognosis after acute myocardial iafarction

1.	Left ventricular ejection fraction
2.	Left ventricular volumes
3.	Infarct perimeter (infarct expansion)
4.	Left ventricular shape (sphericity index)
5.	Wall motion score index
6.	Remote dyssynergy
7.	Hyperkinesia of noninfarcted segments
8.	Infarct zone viability
9.	Mitral regurgitation
10.	Microvascular reperfusion (by MCE)
11.	Early filling deceleration time
12.	E/e′ ratio

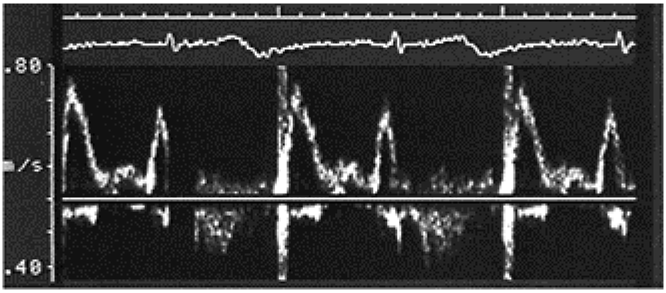
Abbreviations: E/e′: ratio of early transmitral flow velocity to early diastolic velocity of mitral valve annulus; MCE: myocardial contrast echocardiography.

myocardial infarction (Chapter 15),[65,121–123] is associated with consequent left ventricular remodeling, aneurysm formation, development of heart failure, and myocardial rupture.[122,124]

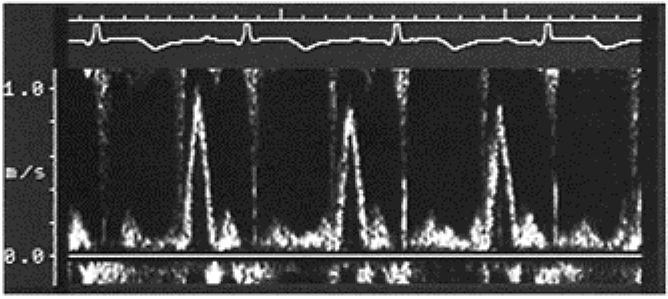
Changing of left ventricular shape toward increased sphericity is associated with worsening of functional status and symptoms of heart failure for 1 year after infarction,[125] although it appears to be less powerful than ejection fraction and the extent of coronary artery disease in predicting poor outcome in patients with coronary artery disease.[125]

Extensive wall motion abnormalities and high wall motion score index are predictors of increased mortality and indicate susceptibility to hypotension, pump failure, severe heart failure, serious dysrhythmia, reinfarction, and cardiogenic shock.[10,18,22,28,59–62,117] Although positive predictive value is less than 50%, these patients should be followed up carefully for development of complications. On the other hand, the presence of a small dysynergic zone is highly suggestive of uncomplicated course.[10,18,22,28,59–62,117] Large wall motion score index at hospital discharge is associated with poor prognosis.[18] Remote dyssynergy indicates multivessel disease and worse prognosis.[28,33]

Hyperkinesia of noninfarcted segments is associated with lower 30-day and 1- and 3-year mortality.[51] Although the frequency of hyperkinesia tends to be lower when echocardiography is performed later in the course of infarction, the prognostic importance of hyperkinesia appears to be unrelated to the time of echocardiographic evaluation.[51]



A



B

Figure 14.5 Examples of normal (or pseudonormal) pattern (A) and restrictive pattern (B) of transmitral flow assessed by pulsed-wave Doppler immediately after admission to coronary care unit.

It has been recently shown that even mild mitral regurgitation detected in the first 48 h in patients with acute myocardial infarction is an independent marker of 1-year mortality.[126]

Lack of infarct zone viability by low-dose dobutamine echocardiography is associated with higher mortality.[103] Effective microvascular reperfusion, as assessed by MCE, predicts functional recovery, prevents left ventricular remodeling,[126–129] and has a beneficial effect on clinical outcome.[130,131]

Among Doppler parameters, it has been demonstrated that short transmitral Doppler deceleration time on day 1 (Figure 14.5) after acute myocardial infarction predicts the development of congestive heart failure better than the ejection fraction.[132] Moreover, restrictive filling has been reported to be the single best predictor of cardiac mortality after myocardial infarction, adding significantly to the predictive power of clinical and echocardiographic markers of systolic dysfunction.[133]

A strong association of short deceleration time on day 3 after infarction and subsequent 6-month left ventricular dilation was reported in a selected group of patients with reperfused acute anterior myocardial infarction.[134] The degree of left ventricular dilation was related to the severity of impairment of left ventricular filling; importantly, a restrictive filling pattern was the most powerful predictor of left ventricular remodeling even after controlling for infarct size.[134] This observation was recently extended to unselected patients after infarction, in whom short early filling deceleration time, as early as on day 1 after infarction, clearly identified those likely to undergo left ventricular remodeling in the following year.[135] Extensive left ventricular remodeling observed in these patients was associated with higher 1- and 5-year mortality (Figure 14.6).[135] It has been recently reported that short deceleration time detected in the early phase of anterior infarction in patients treated with primary angioplasty still retains its prognostic significance even after optimal recanalization of the infarct-related artery and late persistent vessel patency. During a follow-up period of more than 2.5 years, cardiac death was observed only in patients with restrictive filling. They had also more frequent hospital readmissions for heart failure and higher cumulative rates of 2-year mortality and hard cardiac events.[136]

Simple Doppler parameters, such as early filling deceleration time, may have some advantages over left ventricular volumes and ejection fraction in acute settings. Deceleration time was found to be a prognostic parameter superior to ejection fraction after acute myocardial infarction,[133] and it is less time-consuming to measure, highly reproducible, and subject to an almost twofold-lower intraobserver and interobserver variability than end-systolic volume index.[135] Finally, although deceleration time measurement is subject to selection bias, because it is more difficult to measure in the sickest patients with acute myocardial infarction (due to tachycardia, atrial fibrillation, and significant mitral regurgitation), a short initial deceleration time can still identify patients at increased risk of cardiac death, and in whom short- and long-term left ventricular remodeling is likely to occur.[133,135]

Recently, it has been shown that the ratio of the early transmitral flow velocity to the early diastolic velocity of the mitral valve annulus (E/e′) correlates well with mean left ventricular end-diastolic pressure; an E/e′ ratio of >15 was found to be a powerful predictor of survival after acute myocardial infarction, superior to other clinical or echocardiographic features, and providing additional information to these parameters.[137]

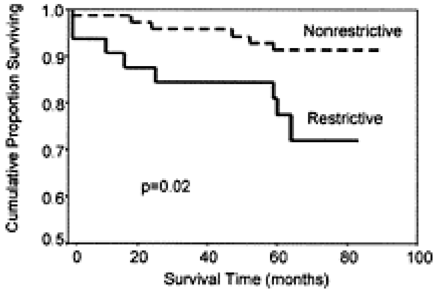


Figure 14.6 Restrictive filling pattern on day 1 is associated with poor 5-year outcome. (Reproduced from P et al. Heart 2001; Otašević P et al. 85:527–32.[135])

Effects of therapy

Evaluation of the natural history[64] of acute myocardial infarction and the effect of reperfusion therapy on systolic and diastolic left ventricular function has revealed that serial echocardiographic studies are useful in detecting changes in left ventricular size, shape, and function in the earliest phase as well as during long-term follow-up. Progressive and complex left ventricular dilation and changes in left ventricular architecture, a process known as remodeling, are clearly associated with infarct expansion in the earliest phase of infarction and large initial left ventricular end-systolic volume (Chapter 15).[65,66]

In patients with successful reperfusion, contractile improvement of initially dysynergic segments could be detected, and the extent of adverse remodeling, was smaller (Figures 14.7 and 14.8).[65,138] Favorable effects on left ventricular volumes, probably through modification

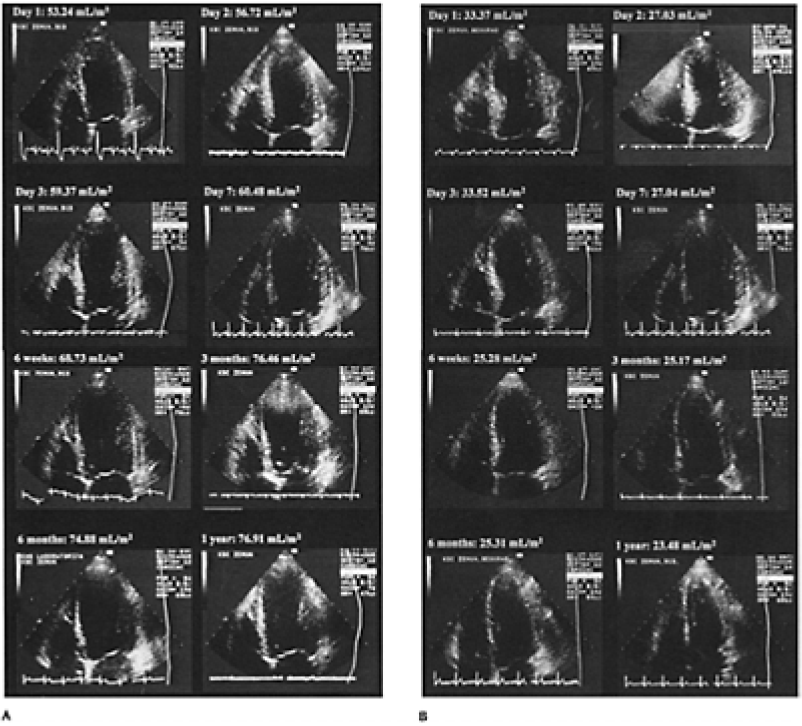


Figure 14.7 (A) Serial two-dimensional echocardiograms in patient with occluded infarct-related artery and adverse left ventricular remodeling. Note increase in left ventricular end-diastolic volumes over time (from 53.24 ml/m^2 on day 1 to 76.91 ml/m^2 after 1 year). (B) In patient with thrombolysis and patent infarct-related artery, left ventricle did not dilate over follow-up (33.37 ml/m^2 on day 1 and 23.48 ml/m^2 after 1 year).

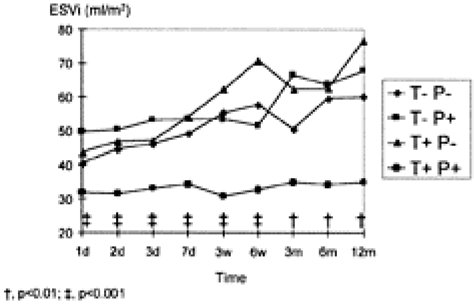


Figure 14.8 Combined impact of thrombolysis (T) and infarct-related artery patency (P) on changes of end-systolic volume index (ESVi) over 1 year after acute myocardial infarction. Note that left ventricular dilation did not occur only in patients who received thrombolysis and who had patent infarct-related artery (T+P+). (Reproduced from Popović AD et al. Am J Cardiol 1996; 77:446–50.[65] © 1996 with permission from Excerpta Medica Inc.)

of distending or deforming forces, have also been demonstrated for pharmacologic treatments such as angiotensinconverting enzyme inhibitors,[139] and intravenous nitroglycerin.[140]

Finally, the viability of the infarct zone clearly indicates therapeutic success.

References

1. Romano S, Dagianti A, Penco M, et al. Usefulness of echocardiography in the prognostic evaluation of non-Q-wave myocardial infarction. Am J Cardiol 2000; 86(Suppl 4A):43G-45G.
2. Premawardhana U, Celermajer DS. Advances in echocardiography. Aust N Z J Med 2000; 30:360–6.
3. Kaul S. Myocardial contrast echocardiography in acute myocardial infarction: time to test for routine clinical use? Heart 1999; 81:2–5.
4. Picano E, Lattanzi F, Orlandini A, et al. Stress echocardiography and the human factor: the importance of being expert. J Am Coll Cardiol 1991; 17:666–9.
5. Czitrom D, Karila-Cohen D, Brochet E, et al. Acute assessment of microvascular perfusion patterns by myocardial contrast echocardiography during myocardial infarction: relation to timing and extent of functional recovery. Heart 1999; 81:12–16.

6. Lepper W, Hoffmann R, Kamp O, et al. Assessment of myocardial reperfusion by intravenous myocardial contrast echocardiography and coronary flow reserve after primary percutaneous transluminal coronary angioplasty in patients with acute myocardial infarction. Circulation 2000; 101:2368–74.
7. Tennant R, Wiggers CJ. The effect of coronary occlusion on myocardial contraction. Am J Physiol 1935; 112:351–61.
8. Arvan S, Varat MA. Two-dimensional echocardiography versus surface electrocardiography for the diagnosis of acute non-Q wave myocardial infarction. Am Heart J 1985; 110:44–9.
9. Loh IK, Charuzi Y, Beeder C, Marshall LA, Ginsburg JH. Early diagnosis of nontransmural myocardial infarction by two-dimensional echocardiography. Am Heart J 1982; 104:963–8.
10. Horowitz RS, Morganroth J, Parroto C, Chen CC, Soffer J, Pauletto FJ. Immediate diagnosis of acute myocardial infarction by two-dimensional echocardiography. Circulation 1982; 65:323–9.
11. Vatner SF. Correlation between acute reductions in myocardial blood flow and function in conscious dogs. Circ Res 1980; 47:201–7.
12. Wyatt HL, Forrester JS, Tyberg JV, et al. Effect of graded reductions in regional coronary perfusion on regional and total cardiac function. Am J Cardiol 1975; 36:185–92.
13. Kerber RE, Marcus ML, Ehrhardt J, et al. Correlation between echocardiographically demonstrated segmental dyskinesis and regional myocardial perfusion. Circulation 1975; 52:1097–114.
14. Lieberman AN, Weiss JL, Jugdutt BI, et al. Two-dimensional echocardiography and infarct size: relationship of regional wall motion and thickening to the extent of myocardial infarction in the dog. Circulation 1981; 63:739–46.
15. Ellis SG, Henschke CI, Sandor T, et al. Relation between the transmural extent of acute myocardial infarction and associated myocardial contractility two weeks after infarction. Am J Cardiol 1985; 55:1412–16.
16. Pandian NG, Skorton DJ, Collins SM. Myocardial infarct size threshold for two-dimensional echocardiographic detection: sensitivity of systolic wall thickening and endocardial motion abnormalities in small versus large infarcts. Am J Cardiol 1985; 55:551–5.
17. Greaves SC. Role of echocardiography in acute coronary syndromes. Heart 2002; 88:419–25.
18. Sabia P, Afrookteh A, Touchstone DA, Keller MW, Esquivel L, Kaul S. Value of regional wall motion abnormality in the emergency room diagnosis of acute myocardial infarction. A prospective study using two-dimensional echocardiography. Circulation 1991; 84(Suppl I):85–92.
19. Gibler WB, Runyon JP, Levy RC, et al. A rapid diagnostic and treatment center for patients with chest pain in the emergency department. Ann Emerg Med 1995; 25:1–8.
20. Peels CH, Visser CA, Kupper AJ, Visser FC, Roos JP. Usefulness of two-dimensional echocardiography for immediate detection of myocardial ischemia in the emergency room. Am J Cardiol 1990; 65:687–91.
21. Heger JJ, Weyman AE, Wann LS, Rogers EW, Dillon JC, Feigenbaum H. Cross-sectional echocardiographic analysis of the extent of left ventricular asynergy in acute myocardial infarction. Circulation 1980; 61:1113–18.
22. Jaarsma W, Visser CA, Eenige van MJ, Verheugt FW, Kupper AJ, Roos JP. Predictive value of two-dimensional echocardiographic and hemodynamic measurements on admission with acute myocardial infarction. J Am Soc Echocardiogr 1988; 1:187–93.
23. Zabalgoitia M, Ismaeil M. Diagnostic and prognostic use of stress echocardiography in acute coronary syndromes including emergency department imaging. Echocardiography 2000; 17:479–93.
24. Cheitlin MD, Armstrong WF, Aurigemma GP, et al. ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography—summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/ASE Committee to Update the 1997 Guidelines for the Clinical Application of Echocardiography). J Am Coll Cardiol 2003; 42:954–70.

25. Dec GW, Waldman H, Southern J, Fallon JT, Hutter AM, Palacios I. Viral myocarditis mimicking acute myocardial infarction. J Am Coll Cardiol 1992; 20:85–9.
26. Narula J, Khaw BA, Dec W, et al. Brief report: recognition of acute myocarditis masquerading as acute myocardial infarction. N Engl J Med 1993; 328:100–4.
27. Sarda L, Colin P, Boccara F, et al. Myocarditis in patients with clinical presentation of myocardial infarction and normal coronary angiograms. J Am Coll Cardiol 2001; 37:786–92.
28. Gibson RS, Bishop HL, Stamm RB, Crampton RS, Beller GA, Martin RP. Value of early two dimensional echocardiography in patients with acute myocardial infarction. Am J Cardiol 1982; 49:1110–19.
29. Kerber RE, Abboud FM. Echocardiographic detection of regional myocardial infarction: an experimental study. Circulation 1973; 47:997–1005.
30. Weiss JL, Bulkley BH, Hutchins GM, Mason SJ. Two-dimensional echocardiographic recognition of myocardial injury in man: comparison with postmortem studies. Circulation 1981; 63:401–8.
31. Nixon JV, Narahara KA, Smitherman TC. Estimation of myocardial involvement in patients with acute myocardial infarction by two-dimensional echocardiography. Circulation 1980; 62:1248–55.
32. Visser CA, Lie KI, Kan G, Meltzer R, Durrer D. Detection and quantification of acute, isolated myocardial infarction by two dimensional echocardiography. Am J Cardiol 1981; 47:1020–5.
33. Stamm RB, Gibson RS, Bishop HL, Carabello BA, Beller GA, Martin RP. Echocardiographic detection of infarct-localized asynergy and remote asynergy during acute myocardial infarction: correlation with the extent of angiographic coronary disease. Circulation 1983; 67:233–44.
34. Weiss JL, Bulkley BH, Hutchins GM, et al. Two-dimensional echocardiographic recognition of myocardial injury in man: comparison with postmortem studies. Circulation 1981; 63:401–8.
35. Nieminen M, Parisi AF, O'Boyle JE, et al. Serial evaluation of myocardial thickening and thinning in acute experimental infarc-tion: identification and quantification using two-dimensional echocardiography. Circulation 1982; 66:174–80.
36. Wyatt HL, Meerbaum S, Heng M, et al. Experimental evaluation of the extent of myocardial dyssynergy and infarct size by two-dimensional echocardiography. Circulation 1981; 63:607–14.
37. Gillam LD, Hogan RD, Foale RA, et al. A comparison of quantitative echocardiographic methods for delineating infarct-induced abnormal wall motion. Circulation 1984; 70:113–22.
38. Buda AJ, Zotz RJ, Pace DP, et al. Comparison of two-dimensional echocardiographic wall motion and wall thickening abnormalities in relation to the myocardium at risk. Am Heart J 1986; 111:587–92.
39. Wyatt HL, Forrester JS, daLuz PL, et al. Functional abnormalities in nonoccluded regions of myocardium after experimental coronary occlusion. Am J Cardiol 1976; 37:366–72.
40. Guth BD, White FC, Gallagher KP, et al. Decreased systolic wall thickening in myocardium adjacent to ischemic zones in conscious swine during brief coronary artery occlusions. Am Heart J 1984; 107:458–64.
41. Lima JAC, Becker LC, Melin JA, et al. Impaired thickening of nonischemic myocardium during acute regional ischemia in the dog. Circulation 1985; 71:1048–59.
42. Jaarsma W, Visser CA, Eenige van MJ, et al. Prognostic implications of regional hyperkinesia and remote asynergy of noninfarcted myocardium. Am J Cardiol 1986; 58:394–8.
43. Stadius ML, Maynard C, Fritz JK, et al. Coronary anatomy and left ventricular function in the first 12 hours of acute myocardial infarction: the Western Washington Randomized Intracoronary Streptokinase Trial. Circulation 1985; 72:292–301.
44. Corya BC, Rasmussen S, Knoebel SB, Feigenbaum H, Black MJ. Echocardiography in acute myocardial infarction. Am J Cardiol 1975; 36:1–10.
45. Nieminen M, Heikkila J. Echoventriculography in acute myocardial infarction. III. Clinical correlations and implication of the noninfarcted myocardium. Am J Cardiol 1976; 38:1–8.

46. Miller TD, Weissler AM, Christian TF, Bailey KR, Gibbons RJ. Quantitative measures of regional asynergy add independent prognostic information to left ventricular ejection fraction in patients with prior myocardial infarction. Am Heart J 1997; 133:640–7.
47. Stack RS, Phillips HR 3rd, Grierson DS, et al. Functional improvement of jeopardized myocardium following intracoronary streptokinase infusion in acute myocardial infarction. J Clin Invest 1983; 72:84–95.
48. Schmidt WG, Sheehan FH, von Essen R, Uebis R, Effert S. Evolution of left ventricular function after intracoronary thrombolysis for acute myocardial infarction. Am J Cardiol 1989; 63:497–502.
49. Schofer J, Lins M, Mathey DG, Sheehan FH. Time course of left ventricular function and coronary patency after saruplase vs Streptokinase in acute myocardial infarction. The PRIMI Trial Study Group. Eur Heart J 1993; 14:958–63.
50. Harrison JK, Califf RM, Woodlief LH, et al. Systolic left ventricular function after reperfusion therapy for acute myocardial infarction. Analysis of determinants of improvement. The TAMI Study Group. Circulation 1993; 87:1531–41.
51. Kjøller E, Køber L, Jørgensen S, Torp-Pedersen C, on behalf of the TRACE Study Group. Long-term prognostic importance of hyperkinesia following acute myocardial infarction. Am J Cardiol 1999; 83:655–9.
52. Grines CL, Topol EJ, Califf RM, et al. Prognostic implications and predictors of enhanced regional wall motion of the noninfarct zone after thrombolysis and angioplasty therapy of acute myocardial infarction. The TAMI Study Groups. Circulation 1989; 80:245–53.
53. Van Reet RE, Quinones MA, Poliner LR, et al. Comparison of two-dimensional echocardiography with gated radionuclide ventriculography in the evaluation of global and regional left ventricular function in acute myocardial infarction. J Am Coll Cardiol 1984; 3:243–52.
54. Kan G, Visser CA, Lie KI, Durrer D. Early two-dimensional echocardiographic measurement of left ventricular ejection fraction in acute myocardial infarction. Eur Heart J 1984; 5:210–17.
55. Visser CA, Kan G, Meltzer RS, et al. Embolic potential of left ventricular thrombus after myocardial infarction: a two-dimensional echocardiographic study of 119 patients. J Am Coll Cardiol 1985; 5:1276–80.
56. Schneider RM, Chu A, Akaishi M, et al. Left ventricular ejection fraction after acute coronary occlusion in conscious dogs: relation to the extent and site of myocardial infarction. Circulation 1985; 72:632–8.
57. Picard MH, Wilkins GT, Gillam LD, Thomas JT, Weyman AE. Immediate regional endocardial surface expansion following coronary occlusion in the canine left ventricle: disproportionate effects of anterior versus inferior ischemia. Am Heart J 1991; 121:753–62.
58. McClements BM, Weyman AE, Newell JB, Picard MH. Echocardiographic determinants of left ventricular ejection fraction after acute myocardial infarction. Am Heart J 2000; 140:284–90.
59. Nishimura RA, Tajik AJ, Shub C, Miller FA, Ilstrup DM, Harrison CE. Role of two-dimensional echocardiography in the prediction of in-hospital complications after acute myocardial infarction. J Am Coll Cardiol 1984; 4:1080–7.
60. Horowitz RS, Morganroth J. Immediate detection of early high risk patients with acute myocardial infarction using two-dimensional echocardiographic evaluation of left ventricular regional wall motion abnormalities. Am Heart J 1982; 103:814–22.
61. Sabia P, Abbott RD, Afrookteh A, Keller MW, Touchstone DA, Kaul S. Importance of two-dimensional echocardiographic assessment of left ventricular systolic function in patients presenting to the emergency room with cardiac-related symptoms. Circulation 1991; 84:1615–24.
62. Peels KH, Visser CA, Dambrink JHE, et al on behalf of the CATS Investigators Group. Left ventricular wall motion score as an early predictor of left ventricular dilation and mortality after first anterior infarction treated with thrombolysis. The CATS investigators group. Am J Cardiol 1996; 77:1149–54.

63. Hammermeister KE, DeRouen TA, Dodge HT. Variables predictive of survival in patients with coronary disease: selection by univariate and multivariate analyses from clinical, electrocardiographic, exercise, arteriographic, and quantitative angiographic evaluations. Circulation 1979; 59:421–30.
64. Picard MH, Wilkins GT, Ray PA, Weyman AE. Natural history of left ventricular size and function after acute myocardial infarction: assessment and prediction by echocardiographic endocardial surface mapping. Circulation 1990; 82:484–94.
65. Popović AD, Nešković AN, Marinković J, Thomas JD. Acute and long-term effects of thrombolysis after anterior wall acute myocardial infarction with serial assessment of infarct expansion and late ventricular remodeling. Am J Cardiol 1996; 77:446–50.
66. Popović AD, Nešković AN, Babić R, et al. Independent impact of thrombolytic therapy and vessel patency on left ventricular dilation after myocardial infarction: serial echocardiographic follow-up. Circulation 1994; 90:800–7.
67. Ito H, Tomooka T, Sakai N, et al. Lack of myocardial reperfusion immediately after successful thrombolysis. Circulation 1992; 85:1699–705.
68. Ito H, Okamura A, Iwakura K, et al. Myocardial perfusion patterns related to thrombolysis in myocardial infarction perfusion grades after coronary angioplasty in patients with acute anterior wall myocardial infarction. Circulation 1996; 93:1993–9.
69. Bolognese L, Antoniucci D, Rovai D, et al. Myocardial contrast echocardiography versus dobutamine echocardiography for predicting functional recovery after acute myocardial infarction treated with primary coronary angioplasty. J Am Coll Cardiol 1996; 28:1677–83.
70. Ito H, Maruyama A, Iwakura K, et al. Clinical implications of the 'no-reflow' phenomenon. A predictor of complications and left ventricular remodeling in reperfused anterior wall myocardial infarction. Circulation 1996; 93:223–8.
71. Sabia PJ, Powers ER, Jayaweera AR, et al. Functional significance of collateral blood flow in patients with recent acute myocardial infarction. A study using myocardial contrast echocardiography. Circulation 1992; 85:2080–9.
72. Agati L, Voci P, Bilotta F, et al. Influence of residual perfusion within the infarct zone on the natural history of left ventricular dysfunction after myocardial infarction: a myocardial contrast echocardiographic study. J Am Coll Cardiol 1994; 24:336–442.
73. Ragosta M, Camarano G, Kaul S, et al. Microvascular integrity indicates myocellular viability in patients with recent myocardial infarction. New insights using myocardial contrast echocardiography. Circulation 1994; 89:2562–9.
74. Ito H, Tomooka T, Sakai N, et al. Time course of functional improvement in stunned myocardium in risk area in patients with reperfused anterior infarction. Circulation 1993; 87:355–62.
75. Czitrom D, Karila-Cohen D, Brochet E, et al. Acute assessment of microvascular perfusion patterns by myocardial contrast echocardiography during myocardial infarction: relation to timing and extent of functional recovery. Heart 1999; 81:12–16.
76. Bolli R. Myocardial 'stunning' in man. Circulation 1992; 86:1671–91.
77. Levine RA, Thomas JD. Insights into the physiologic significance of the mitral inflow velocity pattern. J Am Coll Cardiol 1989; 14:1718–20.
78. Lew WYW. Evaluation of left ventricular diastolic function. Circulation 1989; 79:1393–7.
79. Theroux P, Ross J Jr, Franklin D, Covell JW, Bloor CM, Sasayama S. Regional myocardial function and dimensions early and late after myocardial infarction in the unanesthetized dog. Circ Res 1977; 40:158–65.
80. Pfeffer MA, Pfeffer JM, Fishbein MC, et al. Myocardial infarct size and ventricular function in rats. Circ Res 1979; 44:503–12.
81. Jeremy RW, Hackworthy RA, Bautovich G, Hutton BF, Harris PJ. Infarct artery perfusion and changes in left ventricular volume in the month after acute myocardial infarction. J Am Coll Cardiol 1987; 9:989–95.

82. Fujii J, Yazaki Y, Sawada H, Aizava T, Watanabe H, Kato K. Noninvasive assessment of left and right ventricular filling in myocardial infarction with a two-dimensional Doppler echocardiographic method. J Am Coll Cardiol 1985; 5:1155–60.
83. Choong CY. Diastolic function: its principles and function. In: Weyman AE (ed). Principles and Practice of Echocardiography. Philadelphia: Lea and Febiger, 1994:721–80.
84. Little WC, Ohno M, Kitzman DW, et al. Determination of left ventricular chamber stiffness from the time for deceleration of early left ventricular filling. Circulation 1995; 92:1933–9.
85. Popović AD, Nešković AN, Marinković J, Lee JC, Tan M, Thomas JD. Serial assessment of left ventricular chamber stiffness after acute myocardial infarction. Am J Cardiol 1996; 77:361–4.
86. Chenzbraun A, Keren A, Stern S. Doppler echocardiographic patterns of left ventricular filling in patients early after acute myocardial infarction. Am J Cardiol 1992; 70:711–14.
87. Mojsilović A, Popović AD, Nešković AN, Popović AD. The Wavelet image extension option for analysis and classification of infarcted myocardial tissue. IEEE Trans Biomed Eng 1997; 44:856–66.
88. Picano E, Faletra F, Marini C, et al. Increased echo density of transiently dysynergic myocardium in humans: a novel echocardiographic sign of myocardial ischemia. J Am Coll Cardiol 1993; 21:199–207.
89. Vandenberg BF, Stuhlmuller JE, Rath L, et al. Diagnosis of recent myocardial infarction with quantitative backscatter imaging: preliminary studies. J Am Soc Echocardiogr 1991; 4:10–18.
90. Saeian K, Rhyne TL, Sagar KB. Ultrasonic tissue characterization for diagnosis of acute myocardial infarction in the coronary care unit. Am J Cardiol 1994; 74:1211–15.
91. Soto JR, Beller GA. Clinical benefit of noninvasive viability studies of patients with severe ischemic left ventricular dysfunction. Clin Cardiol 2001; 24:428–34.
92. Braunwald E, Kloner RA. The stunned myocardium: prolonged, postischemic ventricular dysfunction. Circulation 1982; 66:1146–9.
93. Rahimtoola SH. A perspective on the three large multicenter randomized clinical trials of coronary bypass surgery for chronic stable angina. Circulation 1985; 72(Suppl V):V123–35.
94. Pierard LA, De Landsheere CM, Berthe C, Rigo P, Kulbertus HE. Identification of viable myocardium by echocardiography during dobutamine infusion in patients with myocardial infarction after thrombolytic therapy: comparison with positron emission tomography. J Am Coll Cardiol 1990; 15:1021–31.
95. Barillà F, Gheorghiade M, Alam M, Khaja F, Goldstein S. Low-dose dobutamine in patients with acute myocardial infarction identifies viable but not contractile myocardium and predicts the magnitude of improvement in wall motion abnormalities in response to coronary revascularization. Am Heart J 1991; 122:1522–31.
96. Smart SC, Sawada S, Ryan T, et al. Low-dose dobutamine echocardiography detects reversible dysfunction after thrombolytic therapy of acute myocardial infarction. Circulation 1993; 88:405–15.
97. Previtali M, Poli A, Lanzarini L, Fetivau R, Mussini A, Ferrario M. Dobutamine stress echocardiography for assessment of myocardial viability and ischemia in acute myocardial infarction treated with thrombolysis. Am J Cardiol 1993; 72:124G-30G.
98. Salustri A, Elhendy A, Garyfallydis P, et al. Prediction of recovery of ventricular dysfunction after first acute myocardial infarction using low-dose dobutamine echocardiography. Am J Cardiol 1994; 74:853–66.
99. Watada H, Ito H, Oh H, et al. Dobutamine stress echocardiography predicts reversible dysfunction and quantitates the extent of irreversibly damaged myocardium after reperfusion of anterior myocardial infarction. J Am Coll Cardiol 1994; 24:624–30.
100. Iliceto S, Galiuto L, Marchese A, et al. Analysis of microvascular integrity, contractile reserve, and myocardial viability after acute myocardial infarction by dobutamine echocardiography and myocardial contrast echocardiography. Am J Cardiol 1996; 77:441–5.

101. Leclercq F, Messner-Pellenc P, Moragues C, et al. Myocardial viability assessed by dobutamine echocardiography in acute myocardial infarction after successful primary coronary angioplasty. Am J Cardiol 1997; 80:6–10.
102. Barillà F, De Vincentis G, Mangieri E, et al. Recovery of contractility of viable myocardium during inotropic stimulation is not dependent on an increase of myocardial blood flow in the absence of collateral filling. J Am Coll Cardiol 1999; 33:697–704.
103. Bolognese L, Buonamici P, Cerisano G, et al. Early dobutamine echocardiography predicts improvement in regional and global left ventricular function after reperfused acute myocardial infarction without residual stenosis of the infarct related artery. Am Heart J 2000; 139:153–63.
104. Afridi I, Kleiman NS, Raizner AE, Zoghbi WA. Dobutamine echocardiography in myocardial hibernation: optimal dose and accuracy in predicting recovery of ventricular function after coronary revascularization. Circulation 1995; 91:663–70.
105. Maes A, Flameng W, Nuyts J, et al. Histological alterations in chronically hypoperfused myocardium: correlation with PET findings. Circulation 1994; 90:735–45.
106. Bonow RO. Contractile reserve and coronary blood flow in collateral-dependent myocardium. J Am Coll Cardiol 1999; 33:705–7.
107. Kaul S. Response of dysfunctional myocardium to dobutamine. The eyes see what the mind knows! J Am Coll Cardiol 1996; 27:1608–11.
108. Sklenar J, Camarano G, Goodman NC, Ismail S, Kaul S. Dobutamine echocardiography for the determining of the extent of myocardial salvage after reperfusion: an experimental evaluation. Circulation 1994; 90:1503–12.
109. Elhendy A, Trocino G, Salustri A, et al. Low-dose dobutamine echocardiography and rest-redistribution thallium-201 tomography in the assessment of spontaneous recovery of left ventricular function after recent myocardial infarction. Am Heart J 1996; 131:1088–96.
110. Le Feuvre C, Baubian N, Aubry N, Metzger JP, de Vernejoul P, Vacheron A. Assessment of reversible dyssynergic segments after acute myocardial infarction: dobutamine versus thallium-201 single photon emission computed tomography. Am Heart J 1996; 131:668–75.
111. Sicari R, Picano E, Landi P, et al. on behalf of the Echo Dobutamine International Cooperative (EDIC) Study. Prognostic value of dobutamine-atropine stress echocardiography early after acute myocardial infarction. J Am Coll Cardiol 1997; 29:254–60.
112. Carlos ME, Smart SC, Wynsen JC, Sagar KB. Dobutamine stress echocardiography for risk stratification after myocardial infarction. Circulation 1997; 95:1402–10.
113. Bolognese L, Cerisano G, Buonamici P, et al. Influence of infarctzone viability on left ventricular remodeling after acute myocardial infarction. Circulation 1997; 96:3353–9.
114. Nijland F, Kamp O, Verhost PMJ, de Voogt WG, Bosch HG, Visset CA. Myocardial viability: impact on left ventricular dilation after acute myocardial infarction. Heart 2002; 87:17–22.
115. Marini C, Picano E, Varga A, et al. Cyclic variation in myocardial gray level as a marker of viability in man. A videodensitometric study. Eur Heart J 1996; 17:472–9.
116. Nešković AN, Mojsilović A, Jovanović T, et al. Myocardial tissue characterization after acute myocardial infarction using wavelet image decomposition: a novel approach for the detection of myocardial viability in the early postinfarction period. Circulation 1998; 98:634–41.
117. Nishimura RA, Reeder GS, Miller FA, et al. Prognostic value of predischarge 2-dimensional echocardiogram after acute myocardial infarction. Am J Cardiol 1984; 53:429–32.
118. White HD, Norris RM, Brown MA, Brandt PWT, Whitlock RML, Wild CJ. Left ventricular systolic volume as the major determinant of survival after recovery from myocardial infarction. Circulation 1987; 76:44–51.
119. Nešković AN, Otasević P, Bojić M, Popović AD. Association of Killip class on admission and left ventricular dilatation after myocardial infarction: a closer look into an old clinical classification. Am Heart J 1999; 137:361–7.
120. Nešković AN, Marinković J, Bojić M, Popović AD. Predictors of left ventricular thrombus formation and disappearance after anterior wall myocardial infarction. Eur Heart J 1998; 19:908–16.

121. Eaton LW, Weiss JL, Bulkley BH, Garrison JB, Weisfeldt ML. Regional cardiac dilatation after acute myocardial infarction. N Engl J Med 1979; 300:57–62.
122. Schuster EH, Bulkley BH. Expansion of transmural myocardial infarction: a pathophysiologic factor in cardiac rupture. Circulation 1979; 60:1532–8.
123. Picard MH, Wilkins GT, Gillam LD, Thomas JD, Weyman AE. Immediate regional endocardial surface expansion following coronary occlusion in the canine left ventricle: disproportionate effects of anterior versus inferior ischemia. Am Heart J 1991; 121:653–762.
124. Erlebacher JA, Weiss JL, Eaton LW, Kallman C, Weisfeldt ML, Bulkley BH. Late effects of acute infarct dilation on heart size: a two-dimensional echocardiographic study. Am J Cardiol 1982; 49:1120–6.
125. Mancini GB, Bourassa MG, Williamson PR, et al. Prognostic importance of quantitative analysis of coronary cineangiograms. Am J Cardiol 1992; 69:1022–7.
126. Lepper W, Kamp O, Vanoverschelde JL, et al. Intravenous myocardial contrast echocardiography predicts left ventricular remodeling in patients with acute myocardial infarction. J Am Soc Echocardiogr 2002; 15:849–56.
127. Bolognese L, Parodi G, Carrabba N, et al. Impact of microvascular dysfunction on left ventricular remodeling and long-term clinical outcome after primary coronary angioplasty for acute myocardial infarction. Circulation 2004; 109:1121–6.
128. Colonna P, Cadeddu C, Montisci R, et al. Post-infarction microvascular integrity predicts myocardial viability and left ventricular remodeling after primary coronary angioplasty. A study performed with intravenous myocardial contrast echocardiography. Ital Heart J 2002; 3:506–13.
129. Garot P, Pascal O, Simon M, et al. Impact of microvascular integrity and local viability on left ventricular remodeling after reperfused acute myocardial infarction. Heart 2003; 89:393–7.
130. Kenner MD, Zajac EJ, Kondos GT, et al. Ability of the no-reflow phenomenon during acute myocardial infarction to predict left ventricular dysfunction at one-month follow-up. Am J Cardiol 1995; 76:861–8.
131. Sakuma T, Hayashi Y, Sumii K, Imazu M, Yamakido M. Prediction of short- and intermediate-term prognoses of patients with acute myocardial infarction using myocardial contrast echocardiography one day after recanalization. J Am Coll Cardiol 1998; 32:890–7.
132. Poulsen SH, Jensen SE, Gøtzhe O, et al. Evaluation and prognostic significance of left ventricular diastolic function assessed by Doppler echocardiography in the early phase of a first acute myocardial infarction. Eur Heart J 1997; 18:1882–9.
133. Nijland F, Kamp O, Karreman AJP, et al. Prognostic implications of restrictive left ventricular filling in acute myocardial infarction: a serial Doppler echocardiographic study. J Am Coll Cardiol 1997; 30:1618–24.
134. Cerisano G, Bolognese L, Carrabba N, et al. Doppler derived mitral deceleration time: an early strong predictor of left ventricular remodeling after reperfused anterior acute myocardial infarction. Circulation 1999; 99:224–9.
135. Otašević P, Nešković AN, Popović Z, et al. Short early filling deceleration time on day one after acute myocardial infarction is associated with short- and long-term left ventricular remodeling. Heart 2001; 85:527–32.
136. Cerisano G, Bolognese L, Buonamici P, et al. Prognostic implications of restrictive left ventricular filling in reperfused anterior acute myocardial infarction. J Am Coll Cardiol 2001; 37:793–9.
137. Hillis GS, Moller JE, Pellikka PA, et al. Noninvasive estimation of left ventricular filling pressure by E/e′ is a powerful predictor of survival after acute myocardial infarction. J Am Coll Cardiol 2004; 43:360–7.
138. Bolognese L, AN, Parodi G, et al. Left ventricular remodeling after primary coronary angioplasty: patterns of left ventricular dilation and long-term prognostic implications. Circulation 2002; 106:2351–7.

139. St John Sutton M, Pfeffer MA, Plappert T, et al. Quantitative two-dimensional echocardiographic measurements are major predictors of adverse cardiovascular events after acute myocardial infarction. The protective effects of captopril. Circulation 1994; 89:68–75.
140. Jugdutt BI, Warnica JW. Intravenous nitroglycerin therapy to limit myocardial infarct size, expansion, and complications: effect of timing, dosage, and infarct location. Circulation 1988; 78:906–19.

15

Echocardiography in complications of acute myocardial infarction

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Key points

- Echocardiography can accurately detect the cause of new systolic murmurs in patients with acute myocardial infarction.
- Echocardiography is an ideal noninvasive test to screen for mechanical complications of acute myocardial infarction, such as papillary muscle rupture, myocardial free-wall rupture, pseudoaneurysm, and ventricular septal rupture.
- The unique features of echocardiography, its mobility and availability, allow examination of the critically ill at the bedside anywhere in the hospital.
- Echocardiographic examination of unstable patients with ongoing chest pain in the emergency setting is a highly demanding procedure that should not be attempted by the inexperienced.

Two-dimensional and Doppler echocardiography have crucial roles in the detection and assessment of all complications of acute myocardial infarction (Table 15.1). The unique features of echocardiography, its mobility and wide availability, allow the collection of all necessary information at the bedside. The information is often of key importance in decision making in critically ill patients.

Table 15.1 Complications of acute myocardial infarction that may be detected and assessed by echocardiography

1. Acute mitral regurgitation
Papillary muscle dysfunction
Papillary muscle rupture
2. Ventricular septal rupture
3. Free-wall rupture and pseudoaneurysm
4. Infarct expansion, aneurysm, and left ventricular remodeling
5. Left ventricular thrombus
6. Right ventricular infarction
7. Pericardial effusion

Acute mitral regurgitation

Evaluation of the new systolic murmur in patients with acute myocardial infarction may be challenging if limited to clinical examination only. The main efforts are directed to differentiation between acute mitral regurgitation, ventricular septal rupture (see below), or, less frequently, acute tricuspid regurgitation in the setting of right ventricular infarction. The distinction between these potentially catastrophic complications can be easily made in the majority of cases by two-dimensional and Doppler echocardiography. Acute mitral regurgitation in the postinfarction patient may be due to ischemic dysfunction of the papillary muscles and overlying left ventricular wall (papillary muscle region dysynergy), partial papillary muscle rupture, or complete papillary muscle rupture. Two-dimensional echocardiographic imaging suggests etiology and the Doppler techniques assess severity of shunt or regurgitation. Accurate diagnosis strongly influences patient management: in contrast to papillary muscle dysfunction and incomplete rupture, which may be stabilized medically, complete papillary muscle rupture requires emergent cardiac surgery.

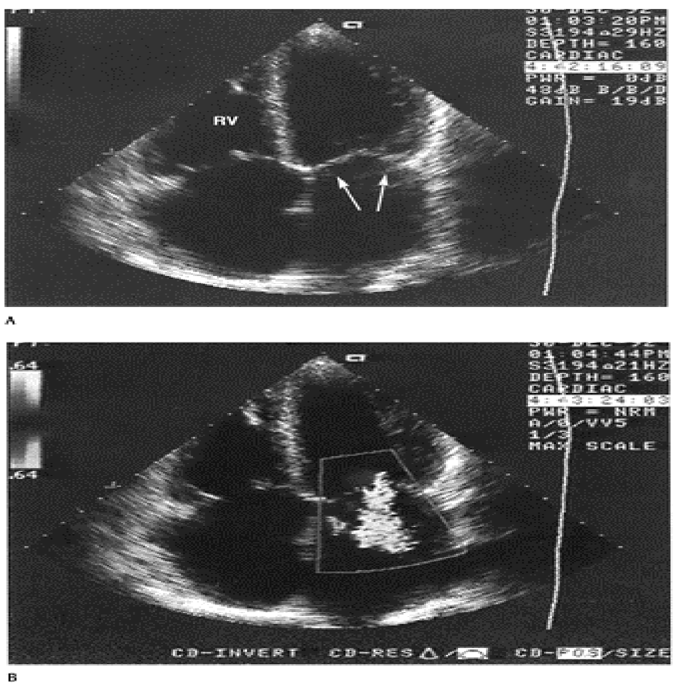
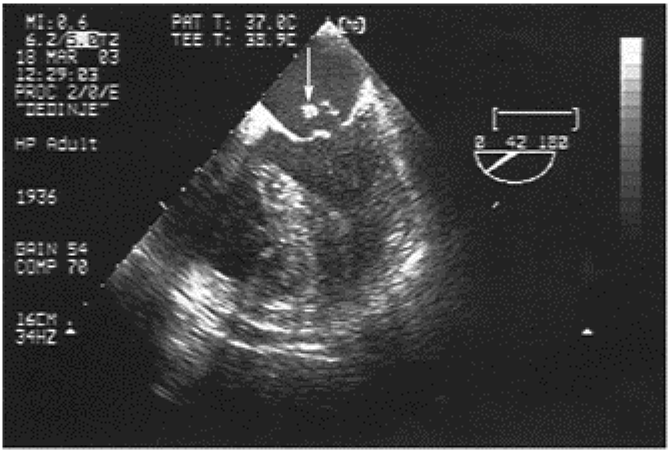


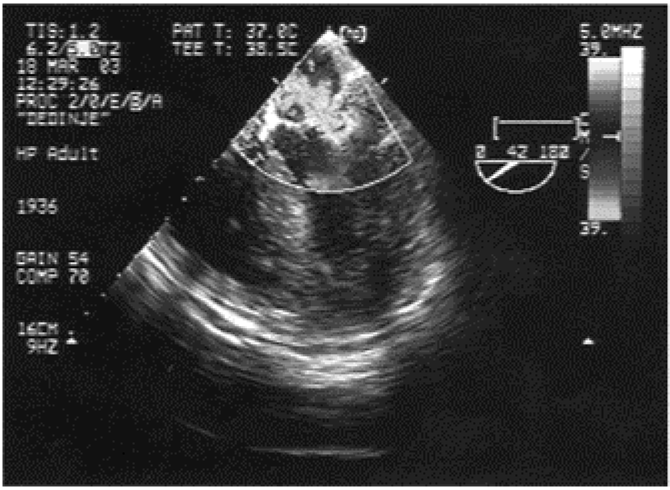
Figure 15.1 Papillary muscle dysfunction in patient with inferior infarction and right ventricular infarction. (A) Two-dimensional echocardiography, four-chamber view.

Note tenting of the mitral valve leaflets (arrows). Right ventricle (RV) appears enlarged. (B) Color Doppler denotes central turbulent jet of moderate mitral regurgitation.

From the echocardiographic point of view, papillary muscle dysfunction can be detected when maximal systolic position of one or both mitral leaflets is displaced toward the left ventricular apex and above the mitral annulus level (Figure 15.1).[1] Color Doppler typically reveals a centrally oriented, turbulent regurgitant jet, although it may be eccentric in some cases due to more pronounced displacement of one of the leaflets. It appears that in addition to ischemic systolic dysfunction of papillary muscles, other mechanisms may play a role in impaired mitral leaflet coaptation, such as left ventricular dilation (especially changes in left ventricular shape toward increased sphericity), mitral annulus dilation, and associated dysynergy of the left ventricular wall underlying the papillary muscle.[2–6] It has been shown that functional mitral regurgitation occurs only when loss of contractility of the papillary muscles is also associated with abnormal wall motion of the underlying myocardium and segments adjacent to the insertion of papillary muscles.[7,8] On two-dimensional echocardiography, complete papillary muscle transaction is typically presented as flailing of either both or one of the mitral leaflets, with an attached echogenic mass that is moving freely during cardiac cycle, and prolapses into the left atrium in systole, moving back into the left ventricle in diastole. The attached mass represents ruptured papillary muscle or its portion (one or more heads) in the case of incomplete rupture, connected by chordal apparatus to the flail leaflets (Figure 15.2). Color Doppler may reveal different jet shapes and directions, and although regurgitation is always severe, it is occasionally difficult to assess its severity by Doppler due to tachycardia and high left atrial pressure. The large regurgitant orifice often results in regurgitation that is more laminar than turbulent and thus more difficult to delineate on color Doppler. Due to dual blood supply from the left anterior descendant and circumflex coronary artery, the anterolateral papillary

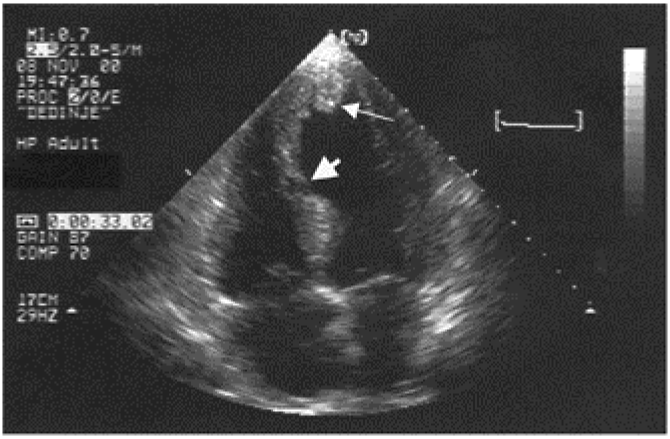


A



B

Figure 15.2 Papillary muscle rupture. (A) Transesophageal echocardiography, longitudinal four-chamber view. Arrow shows flail leaflet with attached mass representing ruptured head of the papillary muscle. (B) Color Doppler reveals torrential mitral regurgitation.



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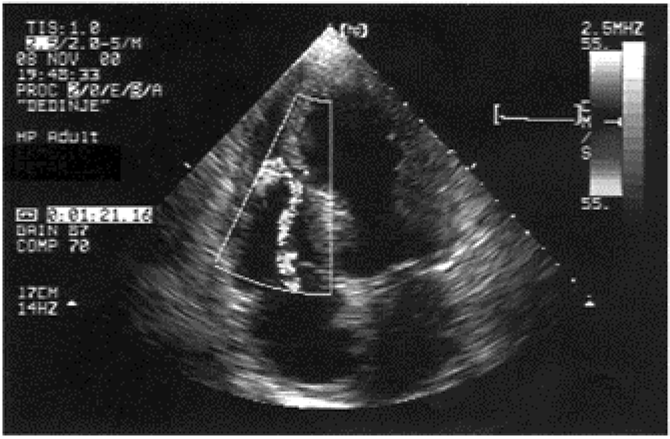

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Figure 15.3 Ventricular septal rupture. (A) Apical four-chamber view. Irregular discontinuity in the midportion of the interventricular septum in patient with anterior infarction, suggesting possible rupture site (thick arrow). In the apex, there is a highly mobile round mass (thin arrow), suggesting thrombus of high embolic potential. (B) Color Doppler shows two turbulent jets of abnormal flow across the rupture site from the

left to the right ventricle. Note that mobile thrombus is not visible in this frame.

muscle is less frequently damaged than the single-vessel-supplied posteromedial papillary muscle.[9] Importantly, in contrast to the ventricular septal rupture that occurs in patients with large infarctions, papillary muscle rupture usually complicates small infarcts.

Mitral regurgitation after myocardial infarction is known to be associated with higher mortality.[10–14] Except in patients with acute severe regurgitation, the reason for increased mortality remains unclear. Since 1933, when Castex reported eight patients with inferior infarction and apical regurgitant systolic murmur consistent with mitral regurgitation,[15] this problem has been extensively studied by various techniques. However, the results of the studies were frequently controversial.[12,13,16–19] These differences may be related to the nature of mitral regurgitation after acute myocardial infarction, which has different etiology, can appear at different time in the course of infarction, can be transient, and can change its severity over time, as well as to the nonuniformity of the entry criteria and the methods for the detection of mitral regurgitation. Recently reported data indicate that early mitral regurgitation after infarction is mild to moderate in most patients, but is associated with features recognized as markers of poor prognosis, such as large infarcts, high

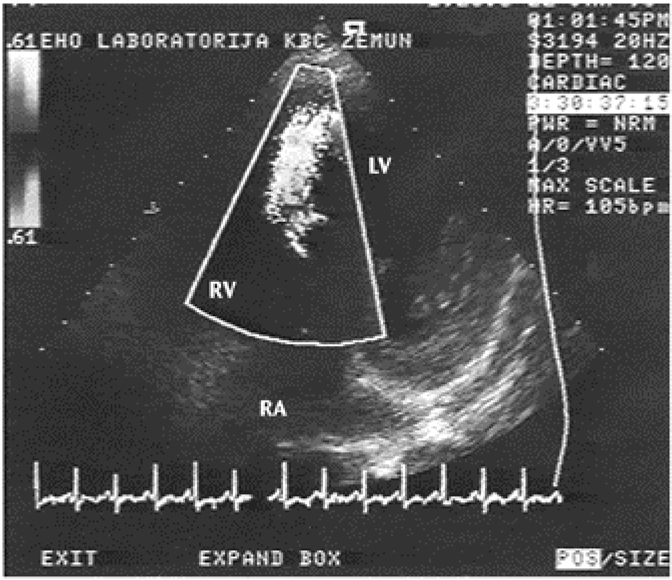


Figure 15.4 Ventricular septal rupture. In modified apical four-chamber view, color Doppler reveals abnormal jet

flow across the apical part of the septum. Width of the jet (9 mm) correlated closely with the diameter of the defect measured intraoperatively. LV: left ventricle; RA: right atrium; RV: right ventricle.

initial end-systolic volume, and older age.[6] On the other hand, comparison of sequential changes of end-diastolic and end-systolic volumes during 1-year follow-up revealed no significant difference in the pattern of left ventricular dilation between patients with and without early mitral regurgitation.[14,20] It seems, therefore, that increased risk and higher mortality in patients with early mitral regurgitation after myocardial infarction are more the consequence of greater left ventricular damage and more severe coronary artery disease than the result of mitral regurgitation itself. It has been recently reported that even mild mitral regurgitation detected within the first 48 h of admission in patients with acute myocardial infarction is a significant independent risk predictor for 1-year all-cause mortality.[21]

Ventricular septal rupture

Ventricular septal defect is a rare complication of acute myocardial infarction in the reperfusion era, with an incidence of 0.2%.[22,23] In clinical practice, it is usually presented as a new holosystolic murmur in patients with acute myocardial infarction that may be difficult to distinguish from the murmur of acute mitral regurgitation (see above). This distinction can be easily made by bedside echocardiography.[24]

In multiple standard and modified echocardiographic views, septal defect can be visualized by two-dimensional echocardiography in 46–100% of cases.[25] The site of the rupture is often localized at the border of the dysynergic zone.[25] Systolic bulging of the ventricular septum toward the right ventricular cavity may be an indirect sign of septal rupture. Acquired defects caused by myocardial necrosis are of irregular shape (Figure 15.3) and complex three-dimensional form. Defects may be serpiginous tunnels through the septal wall, with entry points at different levels on the left and right sides of the septum, and cannot always be detected by two-dimensional echocardiography alone. The defects can also be multiple and small.

Detection of the ventricular septal defect in the region with normal wall motion strongly suggests congenital origin.[26] This may help to avoid the confusion which may arise in individuals with congenital ventricular septal defect and recent myocardial infarction. Doppler techniques greatly improved the diagnostic accuracy of echocardiogra-phy in detection of septal rupture.[25,27] Color Doppler or pulsed-wave Doppler mapping may reveal systolic turbulent flow across the ventricular septum or on the right ventricular side of the septum, suggesting the presence of a communication even in the case of ‘preserved’ continuity of the septum by two-dimensional scan. The maximal width of the turbulent jet by color Doppler (Figure 15.4) may be used to assess

the size of the defect, since the results are in good correlation with intraoperative and autopsy findings.[25]

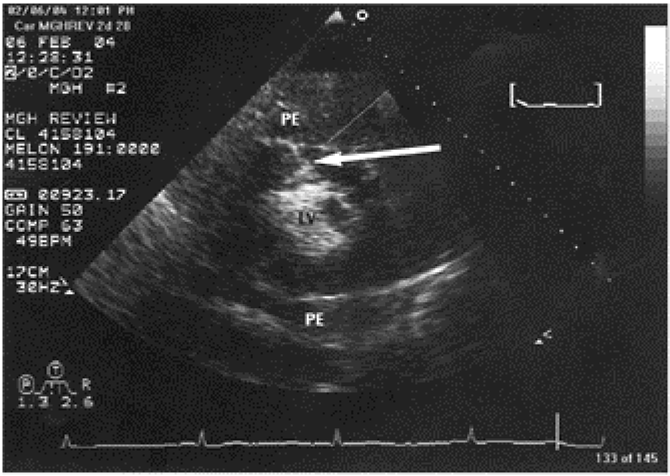


Figure 15.5 Ventricular free-wall rupture. Short-axis parasternal view of the left ventricle of an elderly woman with history of diabetes and hypertension who presented with hypotension and acute myocardial infarction after 3 days of chest pain at home. Pericardial effusion was noted on echocardiogram and intravenous contrast agent was used (filling left ventricular cavity on the figure). Arrow points to the contrast penetrating the myocardium and out to the pericardial space, indicating disruption of the anterolateral myocardial wall. Immediate surgery was performed for repair. LV: left ventricle, PE: pericardial effusion.

In patients with ventricular septal rupture, right ventricular function is crucial in maintaining hemodynamic stability.[28,29] The presence of right-to-left shunt in diastole indicates severe right ventricular dysfunction and is associated with high mortality.[22–25]

Defect of the basal septum due to inferior infarcts tends to have a worse prognosis than the apical defects present in anterior infarcts.[28]

Continuous-wave Doppler can be also used for the assessment of interventricular pressure gradient, and estimation of right ventricular systolic pressure: the lower the gradient, the more severe is the hemodynamic compromise.[25]

Free-wall rupture and pseudoaneurysm

Free-wall rupture

Acute rupture of the left ventricular free wall (Figure 15.5) typically leads to abrupt hemopericardium, cardiac tamponade and sudden death, and electromechanical dissociation, usually before any diagnostic procedure can be performed.[30] Therefore, antemortem diagnosis is rare.[31,32]

Importantly, free-wall rupture does not always result in immediate fatal outcome. Up to 40% may evolve over hours or even days, representing subacute forms of free-wall rupture.[33–36] Subacute rupture is characterized by the presence of moderate-to-severe pericardial effusion, and hypotension of varied severity, with or without signs of tamponade.[35]

Rupture may occur early (within 48 h) or late in the course of infarction. Since mortality is extremely high, a high index of clinical suspicion and timely anticipation may lead to intervention in order to prevent the rupture and/or significantly reduce mortality when it does occur. Hence, some clinical characteristics are associated with increased risk of the rupture, including first transmural infarction in patients of older age, delayed hospital admission, prolonged and recurrent chest pain, acute episodes of arterial hypertension, persistent ST-segment elevation, infarct expansion, and infarct extension.[35–37]

Echocardiography reveals large pericardial effusion in hemodynamically unstable patient with segmental dysynergy due to myocardial infarction, and echocardiographic signs of tamponade (Chapter 2). Rarely, the free-wall defect at the rupture site can be visualized, and flow across thedefect to the pericardial space can be detected by Doppler. Successful use of intravenous contrast echocardiography to exclude or to identify myocardial rupture has been documented.[38] In patients with suspected subacute rupture, administration of a contrast agent can be useful to demonstrate active communication into the pericardium.[39] In addition, it has been recently reported that three-dimensional echocardiography may provide incremental morphologic information in patients with myocardial rupture.[40]

Surgical repair may be life-saving in acute rupture. There are few case reports of successful emergent surgical treatment,[41,42] but the mortality rate remains near 100%. For subacute cases, after prompt initial medical stabilization, surgery should also be considered.

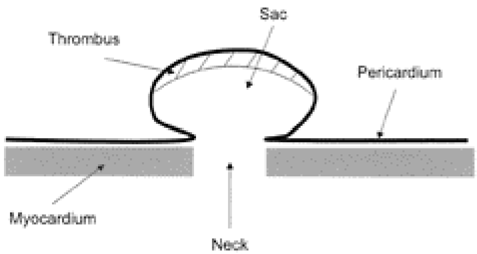
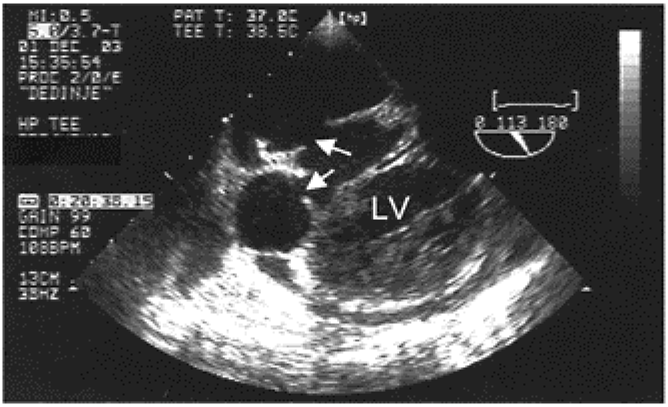


Figure 15.6 Schematic presentation of the pseudoaneurysm. Note the narrow neck; the wall of the pseudoaneurysmal sac consists of the pericardium and laminar thrombus.

Pseudoaneurysm

Patients may survive acute events if myocardial rupture is constrained by pericardial adhesions or thrombosis at the rupture site, leading to pseudoaneurysm formation.[43–45] In contrast to true aneurysm, pseudoaneurysm communicates with the left ventricular cavity by a narrow neck (rupture site), whose diameter is typically at least half that of the maximal diameter of the pseudoaneurysm sack.[44] Through this communication, bidirectional blood flow can be identified by color Doppler during the cardiac cycle. At the margins of the neck of the pseudoaneurysm, sharp discontinuity of the myocardium can be noted, indicating the absence of myocardial layer in the pseudo-aneurysmal sack (Figure 15.6). The pseudoaneurysm wall typically is composed of pericardium and clot.[44] Since pseudoaneurysm requires urgent surgical repair (Figure 15.7) in order to prevent spontaneous rupture that occurs



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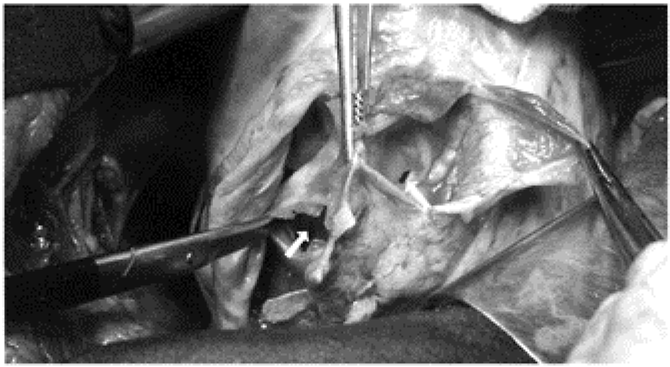

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Figure 15.7 Double pseudoaneurysm. (A) Transesophageal echocardiography: modified longitudinal, two-chamber view. Discontinuity of the inferoposterior myocardial wall at two separate places (arrows), with two narrow-neck aneurysmal sacs, indicating pseudoaneurysm formation. LV: left ventricle. (B) Intraoperative findings: double myocardial rupture (arrows). (Reproduced from Milojević P et al. J Thorac Cardiovasc Surg 2004; 128:765–7.[47] © 2004 with permision

from the American Association for Thoracic Surgery.)

Table 15.2 Echocardiographic features that may be helpful in differentiation of pseudoaneurysm from true aneurysm

	Pseudoaneurysm	*True aneurysm*
Neck:	Narrow	Broad
Wall:	Pericardium	Myocardium, often thin
Margins:	Abrupt myocardial discontinuity	Continuity of myocardial wall

with high incidence,[45–47] it should be promptly distinguished from true aneurysm (Table 15.2), which has a more benign natural history and is approached in a different way.

Infarct expansion, aneurysm formation, and left ventricular remodeling

Infarct expansion

Infarct expansion is defined as acute dilation and thinning of the area of infarction that cannot be explained by additional myocardial necrosis.[48] Infarct expansion begins within the first 24 h after large transmural, mainly anterior infarcts.[49] Patients with infarct expansion have a complicated postinfarction course, and heart failure, aneurysm, and myocardial rupture are more likely to occur.[50–52] Infarct expansion is associated with an early mortality rate as high as 50%,[53] and has an unfavorable impact on late prognosis.[51,54]

Infarct expansion can be recognized by echocardiographic evidence of distortion of ventricular topography as a consequence of elongation of the noncontractile region, causing disproportionate dilation of the infarcted segment, which leads to an increase in the percentage of surface area of the left ventricle occupied by necrotic myocardium.[53,55]

Early echocardiographic studies demonstrated that one-third of patients with myocardial infarction showed infarct expansion during the first few days after admission[53] and that it is associated with late ventricular dilation.[51]

These studies have used the short-axis view of the left ventricle at the papillary muscle level to determine anterior and posterior segment lengths and to define infarct expansion.[53,54] However, since infarct expansion is most common in patients with anterior apical infarcts, this view may not be ideal for the detection of regional changes involving the left ventricular apex. The echocardiographic endocardial surface mapping method allows accurate assessment of infarct expansion and left ventricular dilation;[49–56] however, this method requires optimal imaging in several different planes. Serial measurements of the length of the dysynergic left ventricular endocardium (infarct perimeter) and total left ventricular perimeter, in apical four- and two-chamber views,

allow detection of subtle changes in the infarcted segment (Figure 15.8).[57] Frequent evaluation of the infarct perimeter throughout the early postinfarction phase enables accurate detection of infarct expansion.

Left ventricular aneurysm

Left ventricular aneurysm can be detected in 20–40% of patients after acute myocardial infarction.[58–60] Aneurysm formation is closely related to infarct expansion;[50,51,53,54] consequently, the myocardium in the aneurysmal wall is thin, with gradual transition towards normal myocardial wall at the borders of the aneurysmal sac. Echocardiographic diagnosis of aneurysm can be made if segmental dyskinesis in systole is associated with abnormal segmental geometry in diastole (Figure 15.9). Aneurysm, as well as the time of its formation, strongly influences prognosis. In a series of 158 patients with first myocardial infarction, aneurysm was detected in 22% of patients within 3 months, and in 10% of patients as early as within 5 days.[59] It was detected more frequently in patients with anterior than inferior infarctions (32% vs 9%).[59] Early aneurysm formation was associated with a 1-year mortality rate of 80%, as compared to a 25% mortality rate in those who developed aneurysm later in the course of infarction.[59] Due to stagnant blood flow (smoke sign), left ventricular thrombi can be seen in the aneurysm (Figure 15.10).

Left ventricular remodeling

Infarct expansion is a key event in initiation of the left ventricular remodeling process. This process consists of progressive left ventricular dilation and hypertrophy of noninfarcted segments, leading to changes in left ventricular shape and function over months and years after infarction.[61] The time and magnitude of left ventricular remodeling may be assessed by serial echocardiographic evaluation of left ventricular volume and shape.[62,63]

Although left ventricular remodeling may be prevented or halted by reperfusion therapy,[62] one-third of postinfarction patients may show left ventricular dilation in the

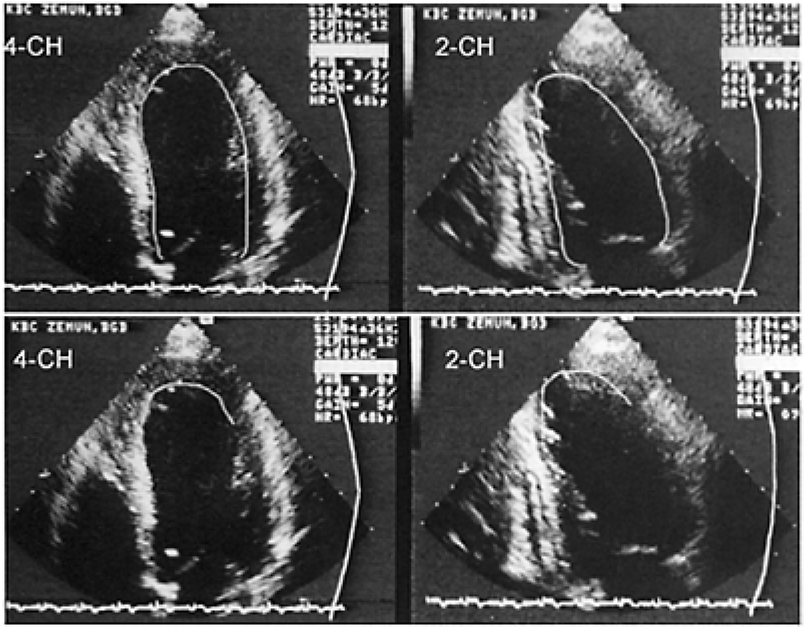

Figure 15.8 Detection of infarct expansion. Tracing of endocardial border in end-diastole in four- and two-chamber views. First, total left ventricular endocardial length is traced and measured in four-chamber and two-chamber (first row of images) views (total left ventricular perimeter). Then, the length of the left ventricular endocardium with wall motion abnormalities (hypokinesia, akinesia, and dyskinesia) is measured (infarct perimeter) in the same frames (bottom right and left images, respectively). Total and infarct perimeters are averaged and mean perimeters are used for calculations. The ratio between two perimeters represents the infarct percentage. Infarct expansion is defined as: (1) an increase of infarct percentage and total perimeter of >5%

on days 2 and 3 in either view; or (2) Initial infarct percentage of >50%, with an increase in total perimeter of >5% on day 2 or 3. (4-CH), Four-chamber view; (2-CH), two-chamber view. (Reproduced from Popović AD et al. Am J Cardiol 1996; 77:446–50[54] with permission from Excerpta Medica, Inc.)

long term despite successful reopening of the infarctrelated artery.[63,64] The remodeling process is heterogeneous, and may be early, late, or progressive; however, in contrast to left ventricular dilation itself, the specific pattern of dilation does not independently affect clinical outcome.[63] Finally, left ventricular remodeling can be predicted by simple demographic and echocardiographic variables.[63–65] High initial left ventricular volumes and wall motion score index are strong predictors of long-term left ventricular dilation.[62–65]

Left ventricular thrombus

The incidence of left ventricular thrombosis is high after acute myocardial infarction.[66–68] The majority of thrombi occur in patients with large anterior infarcts with impaired left ventricular systolic function. The etiologic link between low flow rates, as well as the inverse relationship between flow velocities in the left ventricular apex, and thrombus formation has been recently demonstrated.[69,70] However, thrombi are not always present in such patients, and can also be found in small apical infarcts, with good global systolic function, and, rarely, after inferior infarctions.[67] These facts indicate the complex nature of the process of left ventricular thrombosis and suggest that factors other than infarct size and site may have an impact on the development of left ventricular thrombosis after myocardial infarction.

Two-dimensional echocardiography is a highly accurate noninvasive technique for the detection of left ventricular thrombi, with a sensitivity of 92–95% and a specificity of 86–88%.[71–73] The detection of thrombus is operator-dependent and the success rate is higher if meticulous scanning, by both standardized and nonstandardized, angulated views, is used in suspected cases, with particular efforts to obtain the best possible visualization of the entire left ventricular apex. The use of contrast agents greatly improves the left ventricular thrombus detection rate. The incidence of left ventricular thrombi in anterior infarctions detected by echocardiography is 28–57%,[66,67,72–76] which approaches the incidence reported in autopsy studies of 20–60%.[77,78]

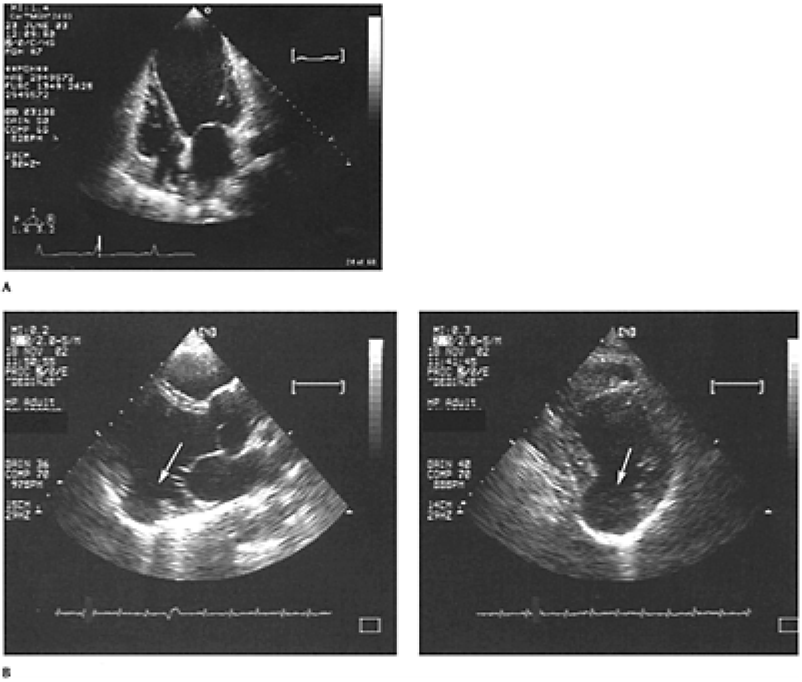

Figure 15.9 True aneurysm. (A) Apical aneurysm: apical four-chamber view. Note abnormal geometry of the apex in diastole. (B) Inferior aneurysm: parasternal long-axis (B.1) and short-axis (B.2) views. Arrows indicate typical broad neck of the aneurysm.

After infarction, thrombus is always located in the dysynergic zone and typically appears as an echogenic mass with a contour distinct from the endocardial border. However, it may be difficult to show a clear demarcation line between the thrombus and underlying myocardium. To distinguish thrombus from the artifacts occasionally seen in the apex, it is helpful to demonstrate the suspected mass in at least two different echocardiographic views.

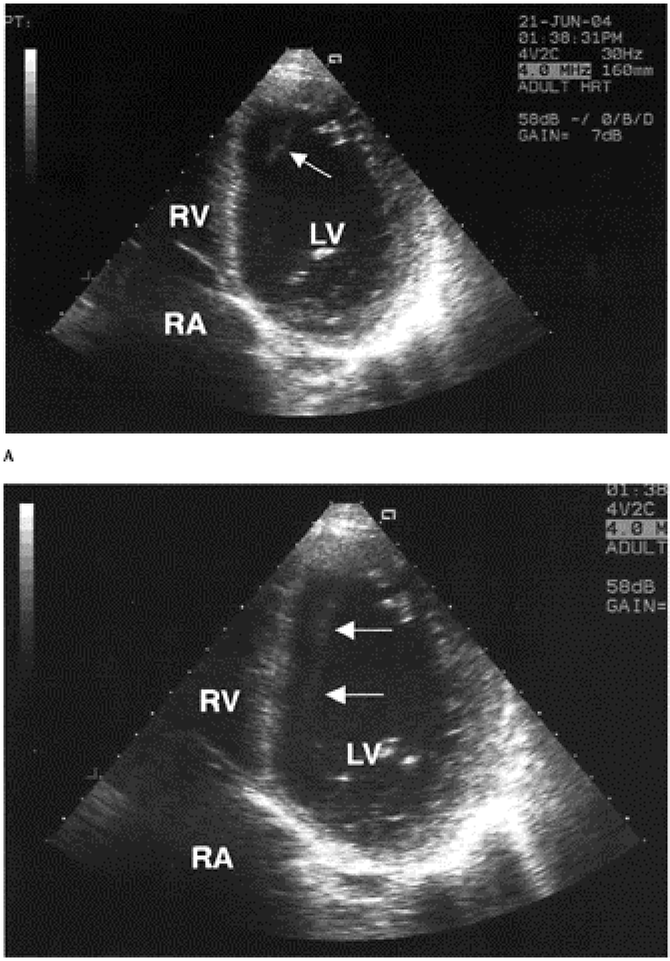

Figure 15.10 Modified apical four-chamber view. Smoke sign (spontaneous echo contrast), indicating stagnant blood flow in the left ventricular cavity after large anterior myocardial infarction. Note the moving smoke-like echoes (arrows) in the left ventricular cavity in different frames (A and B). LV: left ventricle; RA: right atrium; RV: right ventricle.

Transesophageal echocardiography is not always helpful in detection of apical thrombi, because the true left ventricular apex can be missed due to inherent foreshortening of the left ventricular cavity in longitudinal views.

It has been shown that thrombi may appear very early in the course of infarction, as early as within a few hours after experimental infarction.[79] Therefore, frequent echocardiographic examinations are essential to determine the exact incidence of left ventricular thrombosis, particularly in the first week after infarction.[76] Studies that have missed the first week after infarction[80] or have used only a few echocardiographic examinations to detect thrombus may lack substantial data regarding thrombus incidence and natural history.[81,82] In the early phase of formation, thrombi may come out as unclear, cloudy, mobile echoes, before they change into characteristic clot-like appearance.[79] Fresh thrombi tend to have acoustic properties similar to adjacent myocardium in contrast to old, organized thrombi, which may look bright and more echogenic.

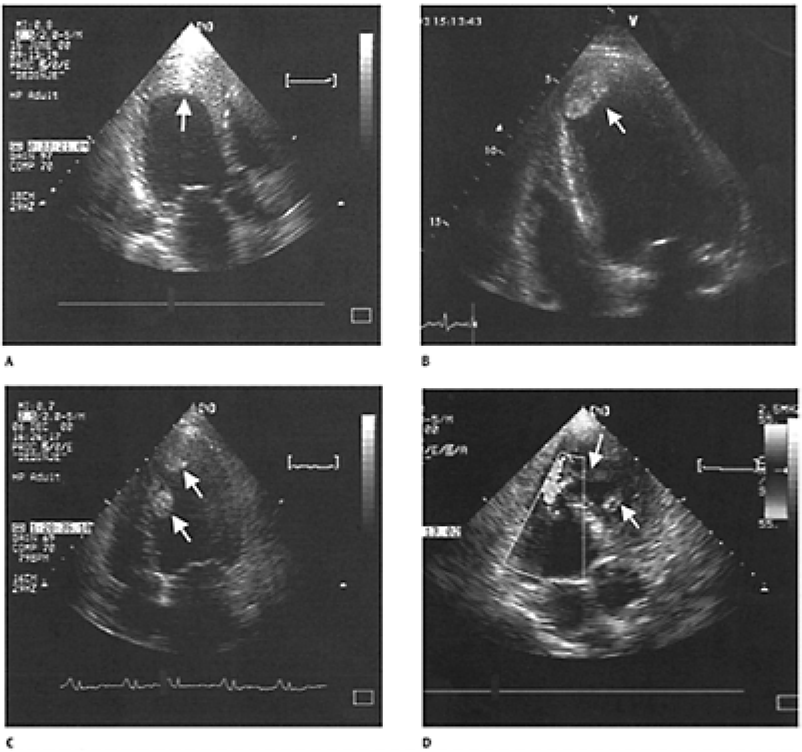

Figure 15.11 Left ventricular thrombus. (A) Huge, laminated, nonprotruding, nonmobile thrombus in the left ventricular apex with low embolic potential (arrow). (B) Prominent, nonmobile thrombus in the

apex (arrow). (C) Highly mobile, large thrombotic mass attached to septal region and apex with significant embolic potential (arrows). (D) In modified four-chamber view of patient with ventricular septal rupture (turbulent jets), mobile masses (arrows), not visible in standard view, can be detected in the apical portion of the left ventricular cavity.

Thrombi may be of various sizes, ranging from very small, hardly detectable echoes to huge masses that can almost obliterate the left ventricular cavity. Thrombi can also be of various shapes. Laminated, nonprotruding thrombi (Figure 15.11 A), without mobile parts, have low embolic potential, in contrast to prominent, globular masses protruding into the left ventricular cavity, and either showing independent mobility or having mobile particles on the surface (Figure 15.11B–D).

There are several clinical implications of left ventricular thrombosis after acute myocardial infarction; therefore, prediction of thrombus formation and resolution may have significant impact on patient management. First, higher mortality has been described in patients with left ventricular thrombi after infarction,[74] especially when these develop within the first 48 h after infarction.[82] Early thrombus formation should be expected in patients with large infarcts, more severe left ventricular dysfunction, more extensive coronary artery disease, and failed infarctrelated artery reperfusion.[76] Since it is well known that these factors are associated with poor prognosis, these findings may explain the higher mortality that has been found in patients with thrombus formation within 48 h after infarction than in those in whom thrombi developed after that period.[83] However, lower early morbidity and mortality and improvement of functional class after 1 year was reported in patients with mural thrombus after infarction.[84] This report explained the favorable impact of the thrombus by an inflammatory process within adjacent myocardium, with consequent healing and thrombus fibrosis that provided mechanical support and reconstruction of the myocardial wall thickness, possibly limiting infarct expansion and remodeling.

The second clinical implication is related to the embolic potential of left ventricular thrombi.[80,84–86] The highest incidence of embolic episodes after infarction is within 12 weeks from an acute event.[67,75] Pooled data showed that arterial embolic events occurred in 18% of patients with left ventricular thrombus detected by echocardiography, but in only 2% of patients without thrombus, and that therisk was especially high in those with mobile and protruding thrombi.[87] Several factors may influence thrombus formation and, therefore, embolic risk. Anticoagulant therapy appears to protect against thrombus formation after acute myocardial infarction.[88] The effect of thrombolysis on thrombus appearance after infarction is controversial; some studies did not demonstrate reduction of thrombus incidence after thrombolysis,[89] while others have shown that thrombolytic therapy, by preservation of the left ventricular function, reduced its incidence.[76,90–93]

Hyperkinesis of the segments of the left ventricular wall adjacent to the thrombus was reported to be associated with increased embolic risk.[85]

Although mitral regurgitation prevents thrombus formation in patients with dilated cardiomyopathy,[94] presumably due to augmented early diastolic flow velocities throughout the entire length of the left ventricle,[94] protecting the left ventricular cavity from a stagnant, thrombogenic blood-flow pattern, no data indicate an association between the presence of mitral regurgitation and left ventricular thrombus formation after acute myocardial infarction.[76]

Initial end-systolic volume, which has been previously reported as a major predictor of survival after acute myocardial infarction,[95] has been identified as the strongest independent predictor of thrombus formation.[76] The fact that lower end-systolic volume has some protective effect against thrombus formation, regardless of the presence of other, thrombus-formation-prone patient characteristics, may facilitate early detection of patients who, among the population with large anterior infarcts, have particularly high probability to develop left ventricular thrombosis. Echocardiographic examination may be repeated if the initial echocardiogram revealed large end-systolic volume, in order not to miss left ventricular thrombosis.

Left ventricular thrombus resolution

Left ventricular thrombi can disappear either spontaneously or after anticoagulation. Thrombus disappearance may be associated with embolic events or may occur without any clinically detectable sign or symptom.[76] The reported rate of thrombus resolution was 20–71%,[66–68,76,79,90,96–99] and it is more likely to occur in patients without left ventricular dyskinesis at the end of the healing phase.[76]

Right ventricular infarction

Right ventricular infarction frequently occurs in association with inferoposterior infarction of the left ventricle, as the consequence of proximal occlusion of the right coronary artery.[100] Involvement of the right ventricular wall can be documented by various diagnostic techniques in 30–50% of these patients, while isolated right ventricular infarctions are rare.

Patients with inferior infarction with right ventricular myocardial involvement are at increased risk of death, shock, and arrhythmia; this appears to be related to the presence of right ventricular myocardial involvement itself rather than the extent of left ventricular myocardial damage.[101]

Accurate diagnosis is essential since the proper management, consisting of volume loading, reperfusion therapy, and, if needed, inotropic support and maintenance of atrioventricular synchrony, should be initiated promptly to avoid severe hemodynamic compromise.[102]

Echocardiography has high sensitivity (82%) and specificity (93%) for the detection of right ventricular infarction.[103,104] Two-dimensional examination reveals various degrees of right ventricular dysynergy and/or right ventricular dilation (Figure 15.12), associated with dysynergy of the inferoposterior segment of the left ventricle.[103–106] Other

echocardiographic signs include paradoxic motion of the ventricular septum,[100] and early opening of

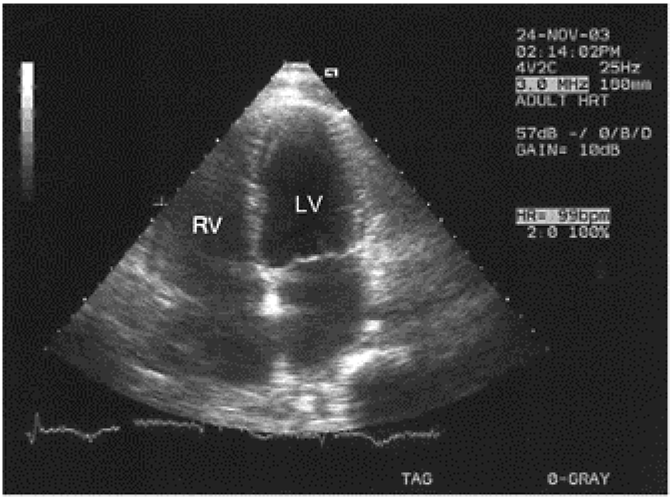

Figure 15.12 Right ventricular infarction. Apical four-chamber view. Right ventricular dilation in acute phase of right ventricular infarction. LV: left ventricle; RV: right ventricle.

the pulmonary valve due to increased right-sided filling pressures.[107]

As a rule, spontaneous complete recovery of right ventricular systolic function can be noted within a few weeks after infarction in a majority of cases, even in the absence of reperfusion.[102,108,109]

Echocardiography may also be helpful in detection of rare right ventricular free-wall rupture, ventricular septal rupture, or right ventricular papillary muscle rupture, all of which may occur after right ventricular infarction.[100,104]

In up to one-third of cases, significant tricuspid regurgitation due to papillary muscle dysfunction, right ventricular dilation, or even papillary muscle rupture is present, and it is associated with increased in-hospital mortality.[110] Like right ventricular dilation and dysynergy, tricuspid regurgitation typically resolves with time.[111] Due to increased right atrial pressure, right-to-left shunt through the patent foramen ovale can also be detected by Doppler or contrast echocardiography.[112,113]

Pericardial effusion

The reported incidence of pericardial effusion in the acute phase of myocardial infarction is 5.6–37%, depending on the techniques used for the detection (M-mode and two-

dimensional echocardiography), and the time of examination after the onset of infarction.[114–120] Studies have shown that pericardial effusion is associated with larger infarctions, anterior infarcts, more extensive wall motion abnormalities, congestive heart failure, severe left ventricular dysfunction, and female gender.[115–119,121]

Other types of pericardial involvement in myocardial infarction patients include free-wall rupture (see above) with tamponade (Chapter 2) and late Dressler's syndrome.

Postinfarction pericardial effusion is usually mild and asymptomatic with little clinical significance.[115–117,120] However, confusion may arise in cases when pericardial friction rub is misinterpreted as cardiac murmur or when chest pain, and/or electrocardiographic changes mimic ongoing myocardial ischemia. In addition, the presence of pericardial effusion after myocardial infarction should always alert the clinician to the possibility of a mild, 'silent', subacute free-wall rupture, without hemodynamic compromise at the moment; then, a high index of suspicion and close follow-up are needed to prevent catastrophe (see above).[36,37]

Two-dimensional echocardiography is an accurate method for the semiquantitative assessment of the quantity of pericardial fluid and allows precise description of its distribution. Echo-free space around the heart due to pericardial effusion should be distinguished from a pericardial fat pad, which is usually located anteriorly, in front of the right ventricular free wall, and typically is not accompanied by signs of posterior effusion. Moreover, distinction from pleural fluid should be made with the

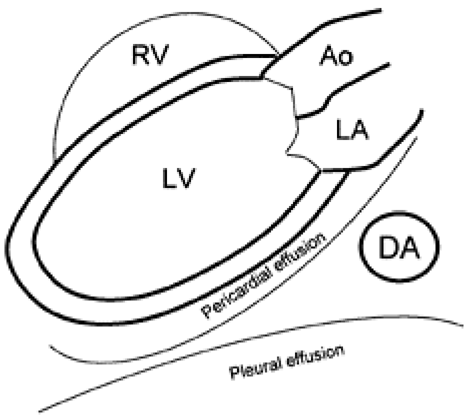

Figure 15.13 Schematic presentation of differentiation between pericardial and pleural effusion. The marker is descending aorta (DA). Pericardial fluid is located anterior to the descending aorta in the long-axis parasternal view, while left pleural effusion is located posteriorly. Ao:

ascending aorta; LA: left atrium; LV: left ventricle; RV: right ventricle.

descending thoracic aorta as a marker: pericardial fluid will appear anterior to the descending aorta in the long-axis parasternal view, and left pleural effusion will be located posterolateral to the descending aorta (Figure 15.13).

References

1. Weyman AE. Left ventricular inflow tract I: the mitral valve. In: Weyman AE (ed.). Principles and Practice of Echocardiography. Philadelphia: Lea and Febiger, 1994:391–470.
2. Kono T, Sabbah HN, Rosman H, et al. Mechanism of functional mitral regurgitation during acute myocardial ischemia. J Am Coll Cardiol 1992; 19:1101–5.
3. Tenenbaum A, Leor J, Motro M, et al. Improved posterbasal segment function after thrombolysis is associated with decreased incidence of significant mitral regurgitation in a first inferior myocardial infarction. J Am Coll Cardiol 1995; 25:1558–63.
4. Kaul S, Spotnitz WD, Glasheen WP, Touchstone DA. Mechanism of ischemic mitral regurgitation. An experimental evaluation. Circulation 1991; 84:2167–80.
5. Kono T, Sabbah HN, Stein PD, Brymer JF, Khaja F. Left ventricular shape as a determinant of functional mitral regurgitation in patients with severe heart failure secondary to either coronary artery disease or idiopathic dilated cardiomyopathy. Am J Cardiol 1991; 68:355–9.
6. Nešković AN, Marinković J, Bojić M, Popović AD. Early predictors of mitral regurgitation after acute myocardial infarction. Am J Cardiol 1999; 84:329–32.
7. Godley RW, Wann LS, Rogers EW, et al. Incomplete mitral leaflet closure in patients with papillary muscle dysfunction. Circulation 1981; 63:565–71.
8. Mittal AK, Langston M, Cohn KE, et al. Combined papillary muscle and left ventricular wall dysfunction as a cause of mitral regurgitation. Circulation 1971; 44:174–80.
9. Nishimura RA, Schaff HV, Shub C, et al. Papillary muscle rupture complicating acute myocardial infarction: analysis of 17 patients. Am J Cardiol 1983; 51:373–7.
10. Heikkila J. Mitral incompetence complicating acute myocardial infarction. Br Heart J 1967; 29:162–9.
11. Maisel AS, Gilpin EA, Klein L, Le Winter M, Henning H, Collins D. The murmur of papillary muscle dysfunction in acute myocardial infarction: clinical features and prognostic implications. Am Heart J 1986; 112:705–11.
12. Lehmann KG, Francis CK, Dodge HT, and the TIMI Study Group. Mitral regurgitation in early myocardial infarction: incidence, clinical detection and prognostic implications. Ann Intern Med 1992; 117:10–17.
13. Tcheng JE, Jackman JD Jr, Nelson CL, et al. Outcome of patients sustaining acute ischemic mitral regurgitation during myocardial infarction. Ann Intern Med 1992; 117:18–24.
14. Nešković AN, Marinković J, Bojić M, Popović AD. Early mitral regurgitation after acute myocardial infarction does not contribute to subsequent left ventricular remodeling. Clin Cardiol 1999; 22:91–4.
15. Castex MR. Les souffles meso-systoliques. Arch Mal Coeur 1933; 26:444.
16. Loperfido F, Biasucci LM, Pennestri F, et al. Pulsed Doppler echocardiographic analysis of mitral regurgitation after myocardial infarction. Am J Cardiol 1986; 58:692–7.
17. Barzilai B, Gessler C Jr, Perez JE, Schaab C, Jaffe AS. Significance of Doppler-detected mitral regurgitation in acute myocardial infarction. Am J Cardiol 1988; 61:220–3.

18. Izumi S, Miyatake K, Beppu S, et al. Mechanism of mitral regurgitation in patients with myocardial infarction: a study using real-time two-dimensional Doppler flow imaging and echocardiography. Circulation 1987; 76:777–85.
19. Leor J, Feinberg MS, Vered Z, et al. Effect of thrombolytic therapy on the evolution of significant mitral regurgitation in patients with a first inferior myocardial infarction. J Am Coll Cardiol 1993; 21:1661–6.
20. Guy TS 4th, Moainie SL, Gorman JH 3rd, et al. Prevention of ischemic mitral regurgitation does not influence the outcome of remodeling after posterolateral myocardial infarction. J Am Coll Cardiol 2004; 43:377–83.
21. Feinberg MS, Schwammenthal E, Shlizerman L, et al. Prognostic significance of mild mitral regurgitation by color Doppler echocardiography in acute myocardial infarction. Am J Cardiol 2000; 86:903–7.
22. Held AC, Cole PL, Lipton B, et al. Rupture of the interventricular septum complicating acute myocardial infarction: a multicenter analysis of clinical findings and outcome. Am Heart J 1988; 116:1330–6.
23. Crenshaw BS, Granger CB, Birnbaum Y, et al. Risk factors, angiographic patterns, and outcomes in patients with ventricular septal defect complicating acute myocardial infarction. Circulation 2000; 101:27–32.
24. Harrison MR, MacPhail B, Gurley JC, et al. Usefulness of color Doppler flow imaging to distinguish ventricular septal defect from acute mitral regurgitation complicating acute myocardial infarction. Am J Cardiol 1989; 64:697–701.
25. Helmcke F, Mahan EF III, Nanda NC, et al. Two-dimensional echocardiography and Doppler color flow mapping in the diagnosis and prognosis of ventricular septal rupture. Circulation 1990; 81:1775–83.
26. Helmcke F, de Souza A, Nanda NC, et al. Two-dimensional and color Doppler assessment of ventricular septal defect of congenital origin. Am J Cardiol 1989; 63:112–16.
27. Panadis IP, Mintz GS, Goel I, McAllister M, Ross J. Acquired ventricular septal defect after myocardial infarction: detection by combined two-dimensional and Doppler echocardiography. Am Heart J 1986; 111:427–9
28. Moore CA, Nygaard TW, Kaiser DL, Cooper AA, Gibson RS. Postinfarction ventricular septal rupture: the importance of location and right ventricular function in determining survival. Circulation 1986; 74:45–55.
29. Radford MJ, Johnson RA, Daggett WM, et al. Ventricular septal rupture: a review of clinical and physiologic features and an analysis of survival. Circulation 1981;64:545–53.
30. Raitt MH, Kraft CD, Gardner CJ, et al. Subacute ventricular free wall rupture complicating myocardial infarction. Am Heart J 1993; 126:946–55.
31. Oliva PB, Hammill SC, Edwards WD, et al. Cardiac rupture, a clinically predictable complication of acute myocardial infarction: report of 70 cases with clinicopathologic correlations. J Am Coll Cardiol 1993; 22:720–6.
32. Spiekerman RC, Brandenburg JT, Avhor RP, et al. The spectrum of coronary heart disease in a community of 30 000. A clinicopathologic study. Circulation 1962; 25:57–65.
33. O'Rourke MF. Subacute heart rupture following myocardial infarction: clinical features of a correctable condition. Lancet 1973; ii: 124–6.
34. Purcaro A, Costantini C, Ciampani N, et al. Diagnostic criteria and management of subacute ventricular free wall rupture complicating acute myocardial infarction. Am J Cardiol 1997; 80:397–405.
35. Figueras J, Cortadellas J, Soler-Soler J. Left ventricular free wall rupture: clinical presentation and management. Heart 2000; 83:499–504.
36. Lopez-Sendon J, Gonzalez A, Lopez de Sa E, et al. Diagnosis of subacute ventricular wall rupture after acute myocardial infarction: sensitivity and specificity of clinical, hemodynamic and echocardiographic criteria. J Am Coll Cardiol 1992; 19: 1145–53.

37. Figueras J, Curos A, Cortadellas J, et al. Relevance of electrocardiographic findings, heart failure, and infarct site in assessing risk and timing of left ventricular free wall rupture during acute myocardial infarction. Am J Cardiol 1995; 76:543–7.
38. Mittle S, Makaryus AN, Mangion J. Role of contrast echocardiography in the assessment of myocardial rupture. Echocardiography 2003; 20:77–81.
39. Garcia-Fernandez MA, Macchioli RO, Moreno PM, et al. Use of contrast echocardiography in the diagnosis of subacute myocardial rupture after myocardial infarction. J Am Soc Echocardiogr 2001; 14:945–7.
40. Puri T, Liu Z, Doddamani S, et al. Three-dimensional echocardiography of post-myocardial infarction cardiac rupture. Echocardiography 2004; 21:279–84.
41. Aravot DJ, Dhalla N, Banner NR, et al. Combined septal perforation and cardiac rupture after myocardial infarction. J Thorac Cardiovasc Surg 1989; 97:815–20.
42. Held P, Dellborg M, Larsson S, et al. Successful repair of extensive inferior myocardial infarction with septal and free wall rupture. Chest 1985; 87:540–1.
43. Frances C, Romero A, Grady D. Left ventricular pseudoaneurysm. J Am Coll Cardiol 1998; 32:557–61.
44. Brown SL, Gropler RJ, Harris KM. Distinguishing left ventricular aneurysm from pseudoaneurysm. Chest 1997; 111:1403–9.
45. Yeo TC, Malouf JF, Oh JK, Seward JB. Clinical profile and outcome in 52 patients with cardiac pseudoaneurysm. Ann Intern Med 1998; 128:299–305.
46. Pretre R, Linka A, Jenni R, Turina M. Surgical treatment of acquired left ventricular pseudoaneurysms. Ann Thorac Surg 2000; 70:553–7.
47. Milojević P, Nešković AN, Vuković M, Nežic D, Djukanović B. Surgical repair of the leaking double postinfarction left ventricular pseudoaneurysm. J Thorac Cardiovasc Surg 2004; 128:765–7.
48. Hutchins GM, Bulkley BH. Infarct expansion versus extension: two different complications of acute myocardial infarction. Am J Cardiol 1978; 41:1127–32.
49. Picard MH, Wilkins GT, Ray PA, Weyman AE. Natural history of left ventricular size and function after acute myocardial infarction: assessment and prediction by echocardiographic endocardial surface mapping. Circulation 1990; 82:484–94.
50. Schuster EH, Bulkley BH. Expansion of transmural infarction: a pathologic factor in cardiac rupture. Circulation 1979; 60:1532–8.
51. Erlebacher JA, Weiss JL, Eaton LW, Kallman C, Weisfeldt ML, Bulkley BH. Late effects of acute infarct dilation on heart size: a two-dimensional echocardiographic study. Am J Cardiol 1982; 49:1120–6.
52. Popović AD, Thomas JD. Detecting and preventing ventricular remodeling after MI. Cleve Clin J Med 1997; 64:319–25.
53. Eaton LW, Weiss JL, Bulkley BH, Garrison JB, Weisfeldt ML. Regional cardiac dilatation after acute myocardial infarction. N Engl J Med 1979; 300:57–62.
54. Erlebacher JA, Weiss JL, Weisfeldt ML, et al. Early dilation of the infarcted segment in acute transmural myocardial infarction: role of infarct expansion in acute left ventricular enlargement. J Am Coll Cardiol 1984; 4:201–8.
55. Weiss JL, Marino PN, Shapiro EP. Myocardial infarct expansion: recognition, significance and pathology. Am J Cardiol 1991; 68:35D-40D.
56. Picard MH, Wilkins GT, Ray P, Weyman AE. Long-term effects of acute thrombolytic therapy on ventricular size and function. Am Heart J 1993; 126:1–10.
57. Popović AD, Nešković AN, Marinković J, Thomas JD. Acute and long-term effects of thrombolysis after anterior wall acute myocardial infarction with serial assessment of infarct expansion and late ventricular remodeling. Am J Cardiol 1996; 77:446–50.
58. Visser CA, Kan G, David GK, Lie KI, Durrer D. Echocardiographic-cineangiographic correlation in detecting left ventricular aneurysm: a prospective study of 422 patients. Am J Cardiol 1982; 50:337–41.

59. Visser CA, Kan G, Meltzer RS, Koolen JJ, Dunning AJ. Incidence, timing and prognostic value of left ventricular aneurysm formation after myocardial infarction: a prospective, serial echocardiographic study of 158 patients. Am J Cardiol 1986; 57:729–32.
60. Matsumoto M, Watanabe F, Goto A, et al. Left ventricular aneurysm and the prediction of left ventricular enlargement studied by two-dimensional echocardiography: Quantitative assessment of aneurysm size in relation to clinical course. Circulation 1985; 72:280–6.
61. McKay RG, Pfeffer MA, Pasternak RC, et al. Left ventricular remodeling after myocardial infarction: a corollary to infarct expansion. Circulation 1986; 74:693–702.
62. Popović AD, Nešković AN, Babić R, et al. Independent impact of thrombolytic therapy and vessel patency on left ventricular dilation after myocardial infarction: serial echocardiographic follow-up. Circulation 1994; 90:800–7.
63. Bolognese L, Nešković AN, Parodi G, et al. Left ventricular remodeling after primary coronary angioplasty: patterns of left ventricular dilation and long-term prognostic implications. Circulation 2002; 106:2351–7.
64. Giannuzzi P, Temporelli PL, Bosimini E, et al. Heterogeneity of left ventricular remodeling after myocardial infarction: results of the Gruppo Italiano per lo Studio della Soprawivenza nell’Infarto Miocardio-3 Echo Substudy. Am Heart J 2001; 141:131–8.
65. Bolognese L, Cerisano G. Early predictors of left ventricular remodeling after acute myocardial infarction. Am Heart J 1999; 138:S79-S83.
66. Asinger RW, Mikell FL, Elsperger J, Hodges M. Incidence of left ventricular thrombosis after acute transmural myocardial infarction: serial evaluation by two-dimensional echocardiography. N Engl J Med 1981; 305:297–302.
67. Weinreich DJ, Burke JF, Pauletto FJ. Left ventricular mural thrombi complicating acute myocardial infarction: long-term follow-up with serial echocardiography. Ann Intern Med 1984; 100:789–94.
68. Keating EC, Gross SA, Schlamowitz RA. Mural thrombi in myocardial infarctions: prospective evaluation by two-dimensional echocardiography. Am J Med 1983; 74:989–95.
69. Maze SS, Kotler MN, Parry WR. Flow characteristics in the dilated left ventricle with thrombus: qualitative and quantitative Doppler analysis. J Am Coll Cardiol 1989; 13:873–81.
70. Delemarre BJ, Visser CA, Bot H, Dunning AJ, with the technical assistance of de Koning H. Prediction of apical thrombus formation in acute myocardial infarction based on left ventricular spatial flow pattern. J Am Coll Cardiol 1990; 15:355–60.
71. Stratton JR, Lighty GW Jr, Pearlman AS, et al. Detection of left ventricular thrombus by two-dimensional echocardiography: sensitivity, specificity, and causes of uncertainty. Circulation 1982; 66:156–66.
72. Visser CA, Kan G, David GK, Lie KI, Durrer D. Two-dimensional echocardiography in the diagnosis of left ventricular thrombus: a prospective study of 67 patients with anatomic validation. Chest 1983; 83:228–32.
73. Pechacek LW, Lazar AV, Sonnemaker RE, et al. Comparison of two-dimensional echocardiography, radionuclide ventriculography and cineangiography in detecting surgically documented left ventricular thrombi. Tex Heart Inst J 1984; 11:118–27.
74. Friedman M, Carlson K, Marcus FI, Woolfenden JM. Clinical correlations in patients with acute myocardial infarction and left ventricular thrombus detected by two-dimensional echocardiography. Am J Med 1982; 72:894–8.
75. Keating EC, Gross SA, Schlamowitz RA, et al. Mural thrombi in myocardial infarctions: prospective evaluation by two-dimensional echocardiography. Am J Med 1983; 74:989–95.
76. Nešković AN, Marinković J, Bojić M, Popović AD. Predictors of left ventricular thrombus formation and disappearance after anterior wall myocardial infarction. Eur Heart J 1998; 19:908–16.
77. Hilden T, Inversen K, Raaschon F, Schwartz M. Anticoagulants in acute myocardial infarction. Lancet 1961; 2:327–31.

78. Veterans Administration Cooperative Study Group. Anticoagulants in acute myocardial infarction: results of cooperative clinical trial. JAMA 1973; 225:724–9.
79. Mikell FL, Asinger RW, Elspherger KJ, Anderson WR, Hodges M. Tissue acoustic properties of fresh ventricular thrombi and visualization by two-dimensional echocardiography: experimental observations. Am J Cardiol 1982; 49:1157–65.
80. Keren A, Goldberg S, Gottlieb S, et al. Natural history of left ventricular thrombi, their appearance and resolution in the posthospitalization period of acute myocardial infarction. J Am Coll Cardiol 1990; 5:790–800.
81. Kontny F, Dale J, Nesvold A, Lem P, Soberg T. Left ventricular thrombosis and arterial embolism in acute anterior myocardial infarction. J Intern Med 1993; 233:139–43.
82. Vecchio C, Chiarella F, Lupi G, Bellotti P, Domenicucci S. Left ventricular thrombus in anterior acute myocardial infarction after thrombolysis. A GISSI-2 connected study. Circulation 1991; 84:512–19.
83. Spirito P, Belloti P, Chiarella F, Domenicucci S, Sementa A, Vecchio C. Prognostic significance and natural history of left ventricular thrombi in patients with acute anterior myocardial infarction: a two-dimensional echocardiographic study. Circulation 1985; 72:774–80.
84. Nihoyannopoulos P, Smith GC, Maseri A, Foale RA. The natural history of left ventricular thrombus in myocardial infarction: a rationale in support of masterly inactivity. J Am Coll Cardiol 1989; 14:903–11.
85. Jugdutt BI, Sivaram CA. Prospective two-dimensional echocardiographic evaluation of left ventricular thrombus and embolism after acute myocardial infarction. J Am Coll Cardiol 1989; 13:554–64.
86. Kupper AJ, Verheught FW, Peels CH, Galema TW, Roos JP. Left ventricular thrombus incidence and behavior studied by serial two-dimensional echocardiography in acute anterior myocardial infarction, ventricular wall motion, systemic embolism and oral anticoagulation. J Am Coll Cardiol 1989; 13:1514–20.
87. Dantzig JMV, Delemarre BJ, Bot H, Visser CA. Left ventricular thrombus in acute myocardial infarction. Eur Heart J 1996; 17:1640–5.
88. Arvan S, Boscha K. Prophylactic anticoagulation for left ventricular thrombi after acute myocardial infarction: a prospective randomized trial. Am Heart J 1987; 113:688–93.
89. Held AC, Gore JM, Paraskos J, et al. Impact of thrombolytic therapy on left ventricular mural thrombi in acute myocardial infarction. Am J Cardiol 1988; 62:310–11.
90. Eigler N, Maurer G, Shah PK. Effect of early systemic thrombolytic therapy on left ventricular mural thrombus formation in acute myocardial infarction. Am J Cardiol 1984; 54:261–3.
91. Bhatnagar SK, al-Yusuf AR. Effects of intravenous recombinant tissue-type plasminogen activator therapy on the incidence and associations of left ventricular thrombus in patients with first acute Q wave anterior myocardial infarction. Am Heart J 1991; 122:1251–6.
92. Lupi G, Domenicucci S, Chiarella F, Bellotti P, Vecchio C. Influence of thrombolytic treatment followed by full dose anticoagulation on the frequency of left ventricular thrombi in acute myocardial infarction. Am J Cardiol 1989; 64:588–90.
93. Natarajan D, Hotchandani RK, Nigam PD. Reduced incidence of left ventricular thrombi with intravenous streptokinase in acute myocardial infarction: prospective evaluation by cross-sectional echocardiography. Int J Cardiol 1988; 20:201–7.
94. Blondheim DS, Jacobs LE, Kotler MN, Costacurta GA, Parry WR. Dilated cardiomyopathy with mitral regurgitation: decreased survival despite a low frequency of left ventricular thrombus. Am Heart J 1991; 122:763–71.
95. White HD, Norris RM, Brown MA, Brandt PWT, Whitlock RML, Wild CJ. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. Circulation 1987; 76:44–51.

96. Visser CA, Kan G, Meltzer RS, Lie KI, Durrer D. Long-term follow-up of left ventricular thrombus after acute myocardial infarction: a two-dimensional echocardiographic study in 96 patients. Chest 1984; 86:532–6.
97. Meltzer RS, Guthaner D, Rakowski H, Popp RL, Martin RP. Diagnosis of left ventricular thrombi by two-dimensional echocardiography. Br Heart J 1979; 42:261–5.
98. Tramarin R, Pozzoli M, Febo O, et al. Two-dimensional echocardiographic assessment of anticoagulant therapy in left ventricular thrombosis early after acute myocardial infarction. Eur Heart J 1986; 7:482–92.
99. Stratton JR, Nemanich JW, Johannessen KA, Resnick AD. Fate of left ventricular thrombi in patients with remote myocardial infarction or idiopathic cardiomyopathy. Circulation 1988; 78:1388–93.
100. Setaro JF, Cabin HS. Right ventricular infarction. Cardiol Clin 1992; 10:69–90.
101. Mehta SR, Eikelboom JW, Natarajan MK, et al. Impact of right ventricular involvement on mortality and morbidity in patients with inferior myocardial infarction. J Am Coll Cardiol 2001; 37:37–43.
102. Haji SA, Movahed A. Right ventricular infarction—diagnosis and treatment. Clin Cardiol 2000; 23:473–82.
103. Bellamy GR, Rasmussen HH, Nasser FN, et al. Value of two dimensional echocardiography, electrocardiography and clinical signs in detecting right ventricular infarction. Am Heart J 1986; 112:304–9.
104. Kozakova M, Palombo C, Distante A. Right ventricular infarction: the role of echocardiography. Echocardiography 2001; 18:701–7.
105. Lopez-Sendon J, Garcia-Fernandez MA, Coma-Canella I, et al. Segmental right ventricular function after acute myocardial infarction: two-dimensional echocardiographic study in 63 patients. Am J Cardiol 1983; 51:390–6.
106. D'Arcy B, Nanda NC. Two-dimensional echocardiographic features of right ventricular infarction. Circulation 1982; 65:167–73.
107. Legrand V, Rigo P. Premature opening of the pulmonary valve in right ventricular infarction. Acta Cardiol 1982; 37:227–31.
108. Goldstein JA. Pathophysiology and clinical management of right heart ischemia. Curr Opin Cardiol 1999; 14:329–39.
109. Ketikoglou DG, Karvounis HI, Papadopoulos CE, et al. Echocardiographic evaluation of spontaneous recovery of right ventricular systolic and diastolic function in patients with acute right ventricular infarction associated with posterior wall left ventricular infarction. Am J Cardiol 2004; 93:911–13.
110. Daubert JC, Langella B, Besson C, et al. Etude prospective des critères diagnostiques et pronostiques de l'atteinte ventriculaire droite a la phase aiguë des infarctus infero-postérieurs. Arch Mal Coeur 1983; 76:991–1003.
111. Descaves C, Daubert J, Langella B, et al. L'insuffisance tricuspidienne des infarctus du myocarde biventriculaire. Arch Mal Coeur 1985; 78:1287–98.
112. Krueger SK, Lappe DL. Right-to-left shunt through patent foramen ovale complicating right ventricular infarction: successful percutaneous catheter closure. Chest 1988; 94:1100–1.
113. Rietveld AP, Merrman L, Essed CE, et al. Right to left shunt with severe hypoxemia, at the atrial level in a patient with hemodynamically important right ventricular infarction. J Am Coll Cardiol 1983; 2:776–9.
114. Wunderink RG. Incidence of pericardial effusion in acute myocardial infarctions. Chest 1984; 85:494–6.
115. Kaplan K, Davison R, Parker M, et al. Frequency of pericardial effusion as determined by M-mode echocardiography in acute myocardial infaction. Am J Cardiol 1985; 55:335–7.
116. Pierard LA, Albert A, Henrard L, et al. Incidence and significance of pericardial effusion in acute myocardial infarction as determined by two-dimensional echocardiography. J Am Coll Cardiol 1986; 8:517–20.

117. Galve E, Garcia-Del-Castillo H, Evangelista A, Batle J, PermanyerMiralda G, Soler-Soler J. Pericardial effusion in the course of myocardial infarction: incidence, natural history, and clinical relevance. Circulation 1986; 73:294–9.
118. Sugiura T, Iwasaka T, Takayama Y, et al. Factors associated with pericardial effusion in acute Q wave myocardial infarction. Circulation 1990; 81(2):477–81.
119. Belkin RN, Mark DB, Aronson L, Szwed H, Califf R, Kisslo J. Pericardial effusion after intravenous recombinant tissue-type plasminogen activator for acute myocardial infarction. Am J Cardiol 1991; 67:496–500.
120. Otasević P, Nešković AN, Bojić M, Popović AD. Pericardial effusion after thrombolysis for acute myocardial infarction: an echocardiographic 1-year follow-up study. Cardiology 1997; 88:544–7.
121. Correale E, Maggioni AP, Romano S, et al. Comparison of frequency, diagnostic and prognostic significance of pericardial involvement in acute myocardial infarction treated with and without thrombolytics. Am J Cardiol 1993; 71:1377–81.

16
Echocardiography in obstruction of native valves

Petar Otašević and Michael H Picard

Key points

- In contrast to prosthetic valves, acute obstruction of native valves occurs rarely.
- Symptoms are nonspecific and depend on the obstruction level.
- Most frequently, symptoms and signs include acute heart failure and/or syncope, while echocardiography provides high diagnostic accuracy.
- Doppler findings in this type of obstruction are similar to those of valve stenosis.

Obstruction of the native valve is a condition that is rarely encountered in clinical practice. It can be the consequence of the growth of cardiac and noncardiac tumors, thrombi, compression of cardiac chambers by extracardiac masses, accessory mitral valve tissue, prolonged left ventricular device support, and miscellaneous other causes. These patients can have new or long-standing murmurs and signs and symptoms of obstruction at different intracardiac levels. Acute obstruction of left-sided valves is most commonly associated with pulmonary edema and/or syncope or right ventricular failure. The role of emergency echocardiography is to distinguish between obstruction of the native valve and other conditions with similar clinical presentation, as well as to suggest possible causes of obstruction. Quantitation of obstruction by continuouswave Doppler is identical to quantitation of pressure gradients in stenotic valves.

Cardiac tumors

Virtually any type of cardiac tumor can cause some type of obstruction of native cardiac valves, and the inflow and/or outflow tracts of both ventricles. We will focus on the most common tumors that cause obstruction.

Myxoma

Myxoma is the most common cardiac tumor in adults; hence, it is the most common cause of obstruction of the native valve. Myxomas are located in the left atrium in over 90% of cases and can cause obstruction of the mitral valve in approximately 10% of patients[1] (Figures 16.1 and 16.2). Reports suggest that symptoms indicative of obstruction of the mitral valve can be found in up to 67% of patients.[2] Myxomas can cause obstruction of the left ventricular outflow tract in approximately 2% of patients,[1]

and there are anecdotal reports of myxomas causing pulmonary stenosis[3] and right ventricular outflow tract obstruction.[4] The therapy of choice is surgical extirpation of the tumor. It is reported that spontaneous regression of myxoma-like obstructive tumors can occur in infants.[5]

Rhabdomyoma

Rhabdomyoma is the most frequent cardiac tumor in infants, but is very rare in adults. Although histologically benign, rhabdomyoma can be functionally malignant if it causes severe obstruction of blood flow.[6] Almost one-quarter of rhabdomyomas cause some degree of

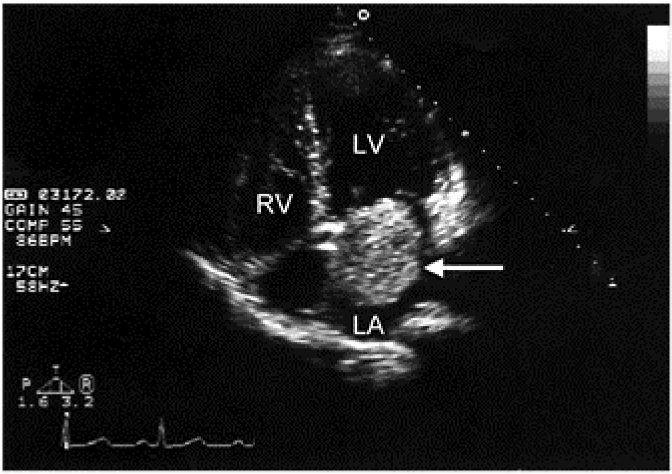

Figure 16.1 Transthoracic apical four-chamber view. Left atrial mass (arrow) consistent with large myxoma obstructs transmitral flow in diastole. LA: left atrium; LV: left ventricle; RV: right ventricle.

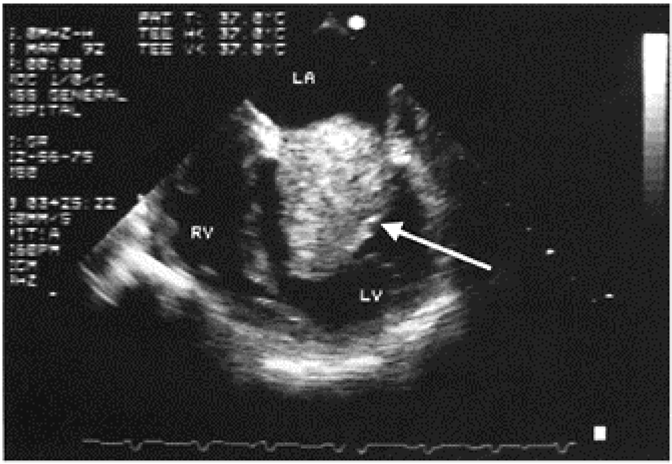

Figure 16.2 Transesophageal longitudinal four-chamber view. Arrow indicates left atrial mass representing large myxoma that causes obstruction of left ventricular inflow in diastole. LA: left atrium; LV: left ventricle; RV: right ventricle.

obstruction.[7] Rhabdomyomas are most commonly located in the left ventricle and the interventricular septum, and usually cause obstruction of the left ventricular outflow tract, although obstruction of the right ventricular outflow tract is not uncommon.[8] Obstruction of atrioventricular valves is rare. The tumor is more likely to be obstructive if it is multiple, and if it is not associated with tuberous sclerosis. Partial or complete spontaneous regression of the tumor occurs in 33.6% of patients, and this is most common in young infants up to 2 years of age.[7] Spontaneous regression does not ocur in adults. Surgical treatment should be reserved for patients with severe obstruction of intracardiac blood flow and/or life-threatening arrhythmia.

Fibroma

Fibromas represent the second most common primary ventricular neoplasm, after rhabdomyoma. They are usually clinically silent, but about 70% of patients eventu

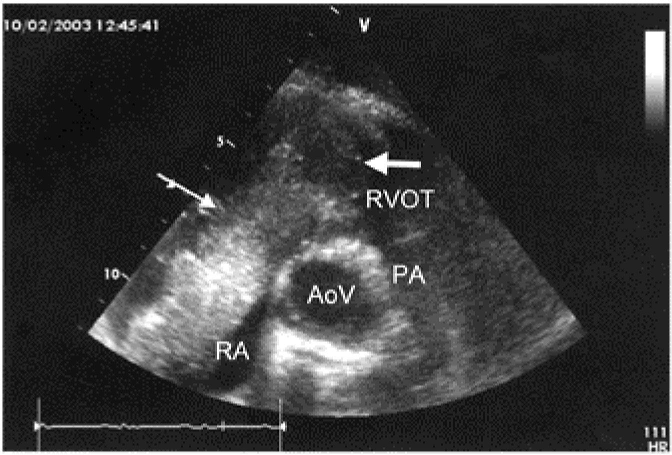

Figure 16.3 Transthoracic short-axis view of the base of the heart. Uterine leiomyoma originating from inferior vena cava obstructs tricuspid valve (thin arrow) and impedes right ventricular filling and right ventricular outflow (thick arrow). AoV: aortic valve; RA: right atrium; RVOT: right ventricular outflow tract; PA: pulmonary artery.

ally develop symptoms, among which signs and symptoms of left ventricular outflow tract obstruction are most common.[9] Obstruction of the right ventricular inflow has also been described.[10] They can be easily distinguished from rhabdomyoma by frequent central calcification.[11]

Sarcoma

Sarcomas comprise up to 75% of primary malignant tumors of the heart in the nonpediatric population. Symptoms are usually rapidly progressive, and prognosis is poor if metastatic dissemination occurs. Sarcomas most frequently originate from the right atrium, and may cause obstruction of the superior or inferior vena cava. These tumors may also obstruct the tricuspid valve, mimicking tricuspid stenosis.[12] Although angiosarcomas are the most frequent sarcomas, it appears that leiomyosarcomas most frequently present with obstruction of blood flow in either the left or right ventricular outflow tract.[13,14] Additionally, leiomyosarcomas and mesenchynomas have been reported to obstruct the mitral inflow and pulmonary veins.[15,16] Surgical resection, if feasible, is the therapy of choice, but, rarely, palliative stenting for pulmonary artery stenosis due to a recurrent primary leiomyosarcoma has been successful.[17]

Noncardiac tumors

Noncardiac tumors are 20–40 times more frequent than cardiac tumors, but rarely cause obstruction of the intracardiac blood flow. Noncardiac tumors can metastasize to the heart through inferior or superior vena (renal cell carcinoma, Wilms' tumor, hepatoma, and uterine leiomyomatosis) and pulmonary veins (bronchial carcinoma), or can directly invade the heart (mediastinal tumors, and bronchial and breast carcinoma). Metastases more frequently involve the pericardium than the myocardium, and are rarely intracavitary. Metastatic sarcoma are usually soft, mobile, and pliable, whereas metastatic carcinoma are solid and fixed and may deform surrounding structures. The most common form of obstruction of blood flow is tricuspid stenosis[18,19] (Figure 16.3), although obstruction of the mitral valve can occur in patients with extension of bronchial carcinoma in the left atrium through pulmonary veins. The treatment is usually palliative.

Thrombi

Thrombi in the left heart are associated with atrial fibrillation and/or extensive wall motion abnormalities caused by myocardial infarction, dilated cardiomyopathy, or Chagas' disease. When symptoms occur, these thrombi typically present as peripheral embolism, although rare cases of giant left atrial thrombus that mimic mitral stenosis ('ball valve obstruction') have been described in patients with atrial fibrillation.[20] Severe aortic stenosis with normal left ventricular systolic function may be associated with free-floating ventricular thrombus,[21] as well as with native aortic valve thrombosis,[22] both of which may cause mild obstruction. Thrombi in the right heart are usually caused by proximal extension of deep vein thrombosis, and frequently present with pulmonary embolism, which is clinically indistinguishable from signs and symptoms of obstruction of right ventricular outflow without emboli in the pulmonary vasculature.

Compression of cardiac chambers by extracardial masses

Most extracardiac masses that may obstruct cardiac chambers originate from either mediastinal tumors (as described in the section on 'Noncardiac tumors') or mediastinal hematoma. An unusual presentation of a mediastinal mass is pulmonary edema caused by obstruction of pulmonary veins.[23] Obstructing pericardial hematomas are most frequent in patients after cardiac surgery and usually present as tamponade or obstruction of the right ventricular outflow tract.[24] Although constrictive pericarditis usually impairs right ventricular filling, it has been reported that constrictive pericarditis may, in extremely rare circumstances, have echocardiographic features of mitral stenosis due to infiltration of the fibrocalcific mass into the base of the posterior mitral leaflet.[25]

Accessory mitral valve tissue

Accessory mitral valve tissue is a rare congenital malformation causing left ventricular outflow tract obstruction.[26] Severe left ventricular outflow tract obstruction is present in over 80% of patients. Surgery is warranted for all patients with severe obstruction, and it has a reported operative mortality rate of 8.9%. Postoperatively, a residual mild gradient across the left ventricular outflow tract was identified in 13.2% of patients, with an additional 13.2% of patients requiring reoperation due to persistent severe obstruction of the left ventricular outflow tract.[27] By reported intraoperative findings, this anomaly is classified as type I-fixed type (A—nodular, B—membranous) and type II-mobile type (A—pedunculated, B—leaflet-like).[28]

Aortic cusp fusion following prolonged left ventricular assist device support

Aortic cusp fusion is not an infrequent complication of prolonged left ventricular assist device support, since it has been reported in about 20% of patients with otherwise normal native valves prior to device implantation.[29] The causes of this phenomenon are not clear. However, aortic cusp fusion may prove to be clinically significant in its creation of a potential source of emboli and infection. Further problems may arise in the case of myocardial recovery, since left ventricular outflow tract obstruction could limit the ability of the heart to be weaned from a left ventricular assist device support.

Miscellaneous causes

Endless case-reports suggest various causes of the obstruction of blood flow. We will mention just a few. A number of reports suggest that cysts (blood cysts and echinococcal cysts) may obstruct either the left ventricular inflow tract or the outflow tract.[30,31] Yolk sac tumor32 and congenital duplication of the tricuspid valve[33] have been shown to impede filling of the right ventricle.

Conclusion

Obstruction of native valves by tumors and thrombi is rare. Left atrial myxoma, the most common cardiac tumor, is the most common cause of obstruction. Echocardiography can assist in the rapid evaluation of symptoms that raise suspicion of obstruction, a situation that is often an acute emergency.

References

1. Keeling IM, Oberwalder P, Anelli-Monti M, et al. Cardiac myxomas: 24 years of experience in 49 patients. Eur J Cardiothorac Surg 2002; 22:971–7.
2. Pinede L, Duhaut P, Loire R. Clinical presentation of left atrial cardiac myxoma. A series of 112 consecutive cases. Medicine (Baltimore) 2001; 80:159–72.
3. Riera JM, Vila IC, Serrano JM, et al. Right ventricular myxoma. A rare case of pulmonary stenosis. Rev Esp Cardiol 1996; 49:153–4.
4. Paraskevaidis IA, Triantafilou K, Karatzas D, Kremastinos DT. Right ventricular multiple myxomas obstructing right ventricular outflow tract. J Thorac Cardiovasc Surg 2003; 126:913–14.
5. Guntheroth WG, Fujioka MC, Reichenbach DD. Spontaneous resolution of obstructive valvular tumors in infants. Am Heart J 2002; 143:868–72.
6. Black MD, Kadletz M, Smallhorn JF, Freedom RM. Cardiac rhabdomyomas and obstructive left heart disease: histologically but not functionally benign. Ann Thorac Surg 1998; 65:1388–90.
7. Verhaaren HA, Vanakker O, De Wolf D, Suys B, François K, Matthys D. Left ventricular outflow obstruction in rhabdomyoma of infancy: meta-analysis of the literature J Pediatr 2003; 143:258–63.
8. Luciani GB, Faggian G, Consolaro G, Graziani S, Martignoni G, Mazzucco A. Pulmonary valve origin of pedunculated rhabdomyoma causing moderate right ventricular outflow obstruction: surgical implications. Int J Cardiol 1993; 41:233–6.
9. Reul GJ, Howell JF, Rubio PA, Peterson PK. Successful partial excision of an intramural fibroma of the left ventricle. Am J Cardiol 1975; 36:262–4.
10. Bapat VN, Varma GG, Hordikar AA, Sivaraman A, Agrawal NB, Tendolkar AG. Right-ventricular fibroma presenting as tricuspid stenosis—a case report. Thorac Cardiovasc Surg 1996; 44:152–4.
11. Geha AS, Weidman WH, Soule EH, McGoon DC. Intramural ventricular cardiac fibroma: successful removal in two cases and review of the literature. Circulation 1967; 36:420–4.
12. Ananthasubramaniam K, Farha A. Primary right atrial angiosarcoma mimicking acute pericarditis, pulmonary embolism, and tricuspid stenosis. Heart 1999; 81:556–8.
13. Ogimoto A, Hamada M, Ohtsuka T, et al. Rapid progression of primary cardiac leiomyosarcoma with obstruction of the left ventricular outflow tract and mitral stenosis. Intern Med 2003; 42:827–30.
14. Willaert W, Claessens P, Shoja A, et al. Ventricular outflow tract obstruction secondary to leiomyosarcoma of the right ventricle. Jpn Heart J 2001; 42:377–86.
15. Gurbuz A, Yetkin U, Yilik L, Ozdemir T, Turk F. A case of leiomyosarcoma originating from pulmonary vein, occluding mitral inflow. Heart Lung 2003; 32:210–14.
16. Peters P, Flachskampf FA, Hauptmann S, Lo HB, Schuster CJ. Bilocular atrial malignant mesenchymoma causing mitral and localized pulmonary vein flow obstruction: diagnosis by transoesophageal echocardiography. Eur Heart J 1992; 13:1585–8.
17. Meckel S, Buitrago-Tellez C, Herrmann R, Jacob AL. Stenting for pulmonary artery stenosis due to a recurrent primary leiomyosarcoma. J Endovasc Ther 2003; 10:141–6.
18. Takeda K, Sawamura S, Tamai H, Hagihara R, Hanaoka K. Reversible tricuspid valve obstruction during removal of renal cell carcinoma with intracardiac tumor extension. Anesth Analg 2000; 91:1137–8.
19. Gunn J, Walker DR, Boyle RM. Malignant thymoma causing tricuspid valve obstruction. Eur Heart J 1990; 11:854–6.
20. Grandmougin D, Letourneau T, Favre JP, Barral X. Paroxysmal postural dyspnea related to a left atrial ball thrombus. Ann Thorac Surg 2002; 74:1691.

21. Davutoglu V, Soydinc S, Celkan A, Kucukdurmaz Z. Left ventricular free-floating ball thrombus complicating aortic valve stenosis. J Heart Valve Dis 2004; 13:197–9.
22. Jobic Y, Provost K, Larlet JM, et al. Intermittent left coronary occlusion caused by native aortic valve thrombosis in a patient with protein S deficiency. J Am Soc Echocardiogr 1999; 12:1114–16.
23. Eicher JC, de Nadai L, Falcon-Eicher S, et al. Unexplained pulmonary edema: demonstration of obstruction to pulmonary venous return by transesophageal echocardiography: Apropos of 4 cases. Arch Mal Coeur Vaiss 1997; 90:67–74.
24. Tardif JC, Taylor K, Pandian NG, Schwartz S, Rastegar H. Right ventricular outflow tract and pulmonary artery obstruction by post-operative mediastinal hematoma: delineation by multiplane transesophageal echocardiography. J Am Soc Echocardiogr 1994; 7:400–4.
25. Pai RG, Tarazi R, Wong S. Constrictive pericarditis causing extrinsic mitral stenosis and a left heart mass. Clin Cardiol 1996; 19:517–19.
26. Ascuitto RJ, Ross-Ascuitto NT, Kopf GS, Kleinman CS, Talner NS. Accessory mitral valve tissue causing left ventricular outflow obstruction (two-dimensional echocardiographic diagnosis and surgical approach). Ann Thorac Surg 1986; 42:581–4.
27. Prifti E, Bonacchi M, Bartolozzi F, Frati G, Leacche M, Vanini V. Postoperative outcome in patients with accessory mitral valve tissue. Med Sci Monit 2003; 9:RA126–33.
28. Prifti E, Frati G, Bonacchi M, Vanini V, Chauvaud S. Accessory mitral valve tissue causing left ventricular outflow tract obstruction: case reports and literature review. J Heart Valve Dis 2001; 10:774–8.
29. Baradarian S, Dembitsky WP, Jaski B, et al. Left ventricular outflow tract obstruction associated with chronic ventricular assist device support. ASAIO J 2002; 48:665–7.
30. Minneci C, Casolo G, Popoff G, Sulla A, Comin CE, Pedemonti E. A rare case of left ventricular outflow obstruction. Eur J Echocardiogr 2004; 5:72–5.
31. Shinada K, Etsuda H, Miyamoto A, Watanabe T, Sawada H. Cystic mass in the left ventricular outflow tract in a 76-year-old man. J Cardiol 2004; 43:193–6.
32. Morin MJ, Hopkins RA, Ferguson WS, Ziegler JW. Intracardiac yolk sac tumor and dysrhythmia as an etiology of pediatric syncope. Pediatrics 2004; 113:e374–6.
33. Bisognano JD, Merrick DT, Popylisen SL, Hoffman MS, Weinberger HD. Congenital duplication of the tricuspid valve presenting as a circumscribed obstructive mass in the right ventricular outflow tract. J Am Soc Echocardiogr 1998; 11:480–2.

17
Emergency echocardiography in the patient with a prosthetic valve

Mauro Pepi and Frank A Flachskampf

Key points

- Suspected malfunction or endocarditis of a prosthetic valve should prompt a transesophageal examination.
- Obstruction and regurgitation may coexist, as when the occluder is thrombotically immobilized.
- If there is suspicion of obstruction in a mechanical valve, the occluder opening should be closely scrutinized, including examination by fluoroscopy, and thrombus should be sought.
- Sudden new regurgitation can be due to structural deterioration, endocarditis, or thrombosis.

Several clinical situations due to a diseased or malfunctioning prosthetic valve constitute clinical emergencies. These scenarios often arise suddenly, are life-threatening, and require immediate firm diagnosis for therapeutic decisions, which more often than not include valve surgery. Besides the clinical evaluation, echo has a dominant role in the work-up of these patients. Valve prosthesis-related emergencies may be due to prosthetic obstruction of varying degree and acuity, prosthetic regurgitation of varying degree and acuity, infective endocarditis, embolism, or a combination of these.

Prosthetic valve thrombosis

Prosthetic valve thrombosis remains a constant source of postoperative morbidity and mortality. Despite recent advances in diagnostic methods, obstructive and non-obstructive prosthetic valve thrombosis continues to be a challenge.[1–3]

Thrombosis occurs almost exclusively in mechanical valves, leading to predominant stenosis with or without some regurgitation depending on the dynamic of the prosthetic leaflet(s). The onset of symptoms may be very variable, gradual, or sudden. In fact, symptoms and clinical findings depend on how rapidly the prosthetic obstruction develops and on the severity of obstruction. Symptoms include dyspnea, generalized weakness, or, in cases of rapid progression of obstruction pulmonary edema or shock, cerebral as well as coronary or peripheral embolization.

The incidence of prosthetic valve thrombosis is twice as high in the mitral as in the aortic position, and it is even higher in the tricuspid position, independently of the

prosthesis type. Echocardiography should focus on determining movement of the occluder(s), and Doppler gradients across the valve. Moreover, mechanisms determining obstruction (thrombus, pannus, or both) may be identified as well as the hemodynamic consequences of obstruction (left ventricular function, right ventricular function, and pulmonary systolic pressure).[3–6]

In the presence of clinical suspicion of prosthetic valve thrombosis, transthoracic echocardiography should be immediately performed, and transesophageal echocardiography (TEE) and other diagnostic tests (including cinefluoroscopy) are generally recommended after this first step approach. However, in cardiogenic shock and ventilated patients, TEE should be immediately performed.

Doppler gradients

Prosthetic valve obstruction may be suspected if the Doppler-derived gradients are twice as high as those given for a normal prosthetic model of the same outer diameter

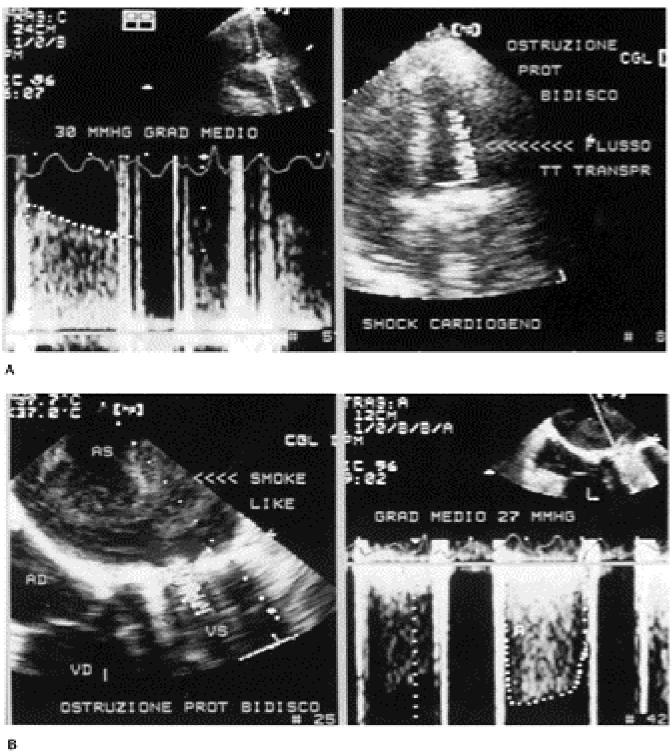

Figure 17.1 Cardiogenic shock in a patient with a bileaflet prosthetic valve in the mitral position. Top panel left:

transthoracic continuous-wave Doppler showing a very high mean gradient across the prosthesis (30 mmHg with a typical rectangular shape pattern). Top panel right: transthoracic apical four-chamber view: a very thin and turbulent jet is recorded across the valve. Bottom panels left and right: transesophageal four-chamber view and continuous-wave Doppler from this view confirm the transthoracic data. TEE also shows marked smoke (spontaneous echo contrast) in the left atrium.

and with comparable transprosthetic flow conditions. Therefore, tables with normal values of different prosthetic model may be very useful in comparing gradients in individual cases, but due to high variability in gradients, particularly in the aortic position, it is more important to evaluate and compare (when available) gradients obtained in acute conditions with previous Doppler gradients from the same patient. It is therefore very important to recommend Doppler echocardiographic follow-up in the early and late postoperative periods to facilitate recognition of changes in Doppler gradients. Although an increased mean gradient is the hallmark of prosthetic valve thrombosis, approximately up to 20% of cases of mitral prosthetic valve thrombosis (excluding those with nonobstructive thrombosis) have a normal Doppler gradient at rest.[7,8] This occurs mainly or exclusively in patients with bileaflet prostheses in the mitral position; therefore, inspection of disk motion by transthoracic echocardiography, TEE, or cinefluoroscopy is mandatory. In patients with aortic prostheses, we may face the opposite situation: due to the well-known pressurerecovery phenomenon (overestimation of Doppler gradient when compared to invasive true pressure drop due to distal pressure recovery), we may observe very high gradients, particularly in cases with small size models even in normally functioning prostheses.[4,9,10]

The continuity equation can be used to estimate mitral and aortic valve areas, and pressure half-time may be used to calculate tricuspid and mitral valve areas. Note, however, that pressure half-times depend also on prosthetic valve type and should be compared with early normal postoperative values presumably reflecting normal prosthetic function. The continuity equation is particularly useful when transvalvular flow is decreased, as in patients with left ventricular dysfunction.

For assessment of all these data in acute conditions, it is very important to evaluate from the parasternal window the basic linear measurements, including the left ventricular outflow tract (to be inserted into the continuity equation), and to measure gradients of the mitral and aortic prostheses in the four- and five-chamber apical views. From these

views, abnormal flow jets (very thin and turbulent inflow jet(s) for the obstructed mitral valve and/or abnormal regurgitant jets for the mitral and aortic prostheses) should be researched. All these data may be also easily obtained by the transesophageal approach, and in this regard transthoracic parameters may facilitate a focused and goal-oriented transesophageal study to visualize prosthetic jets to confirm or complete the diagnosis. For regurgitation, the transthoracic approach is very useful to evaluate the severity of the regurgitant lesion for aortic prostheses, and TEE may further improve

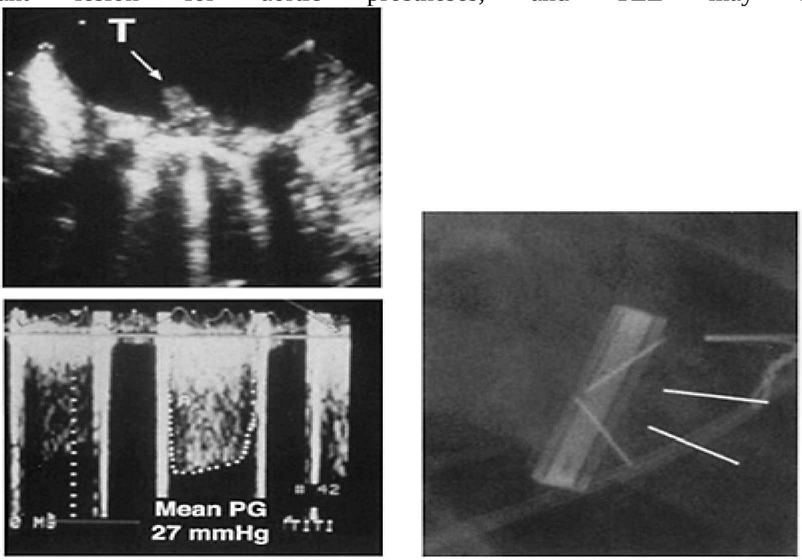

Figure 17.2 Patient in NYHA functional class III with a bileaflet mitral prosthesis: top panel left: TEE shows a large thrombus (T) on the atrial side of the prosthesis (arrow). Bottom panel left: a markedly elevated mean gradient is recorded by continuous-wave Doppler (27 mmHg, four-chamber TEE view) demonstrating extreme obstruction of the valve. Right panel: cinefluoroscopy in the same patient demonstrates severe reduction of leaflet motion. The two white lines indicate the expected normal opening angles of the two leaflets.

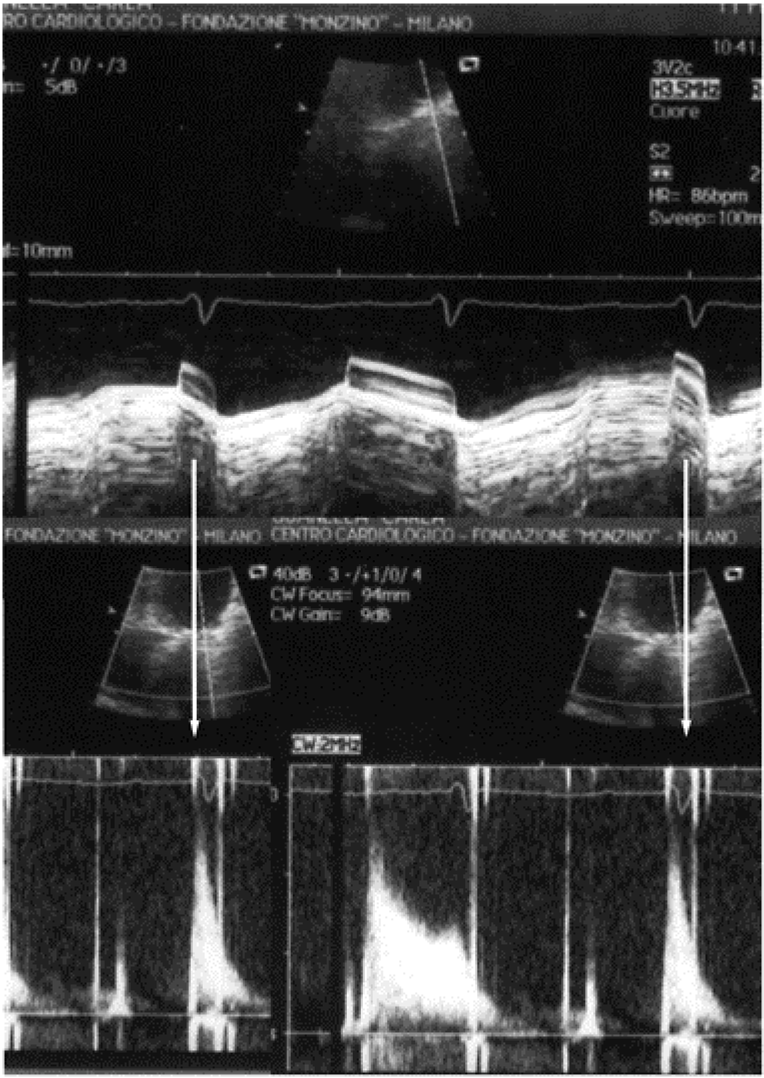

Figure 17.3 Patient with a single-leaflet mitral prosthesis in NYHA functional class II. M-mode from the transthoracic four-chamber view (top panel) and continuous-wave Doppler in the same view (bottom panel) allow identification of abnormal, intermittent disk opening and altered flow across the valve. During regular sinus rhythm,

the disk opens only at end-diastole of every second beat. Arrows indicate the temporal correspondence between disk opening and flow across the valve at end-diastole.

recognition of the site of the regurgitant jet (intra- or paraprosthetic), while the site and severity of mitral valve prosthetic jets, are easily assessed by transesophageal studies (see also section on 'Prosthetic regurgitation' below).[5,6,11–13]

Figure 17.1 shows an example of Doppler transthoracic and transesophageal studies in a patient with prosthetic valve obstruction in the mitral position. The extreme increase in mean transprosthetic gradient clearly indicates severe obstruction.

Disk motion and excursion

A reduced or absent leaflet motion associated with an increased pressure gradient is the hallmark of prosthetic valve thrombosis. Cinefluoroscopy is better suited than echocardiography to evaluate precisely the actual valve opening and closing angles. However, thanks to recent advances in transducers and ultrasound units, transthoracic echocardiography and TEE now allow visualization of disk motion in a large number of cases. Typically, in multiple cross-sectional views (from the parasternal and apical views, including off-axis views), we may observe persistent restriction of prosthetic disk opening by M-mode and two-dimensional (2-D) echo. Altered mobility may involve one or both of the disks in bileaflet models (with different levels of alteration—from stuck leaflet to mild alteration of disk excursion) or the single occluder in single disk (or ball) prosthesis. Even with recent transthoracic improvements in quality, TEE is significantly more accurate in detecting occluder motion, particularly in the mitral position. Disk motion should be evaluated carefully throughout the studies, since it is not uncommon to observe cases with intermittent restriction of prosthetic disk opening. In these cases, M-mode, 2-D, and Doppler may detect varying degrees and delays in the opening of the disk and associated intermittent increase of transprosthetic gradients.

Figures 17.2 and 17.3 show examples of a persistent and an intermittent disk restriction in two patients with a bileaflet prosthesis in the mitral position.

Prosthetic valve thrombus and pannus

TEE is a sensitive and accurate tool for diagnosing prosthetic valve thrombosis, including the characteristics and mechanisms producing obstruction. Moreover, in cases with embolism without clinical and echocardiographic signs of obstruction, TEE allows detection of nonobstructive thrombosis. Thrombus is defined as a distinct mass of abnormal echoes attached to the prosthesis and clearly seen throughout the cardiac study. The site, size, motility, and echostructure of the mass should be annotated. Through multiple views and multiplane angle rotation, the extension and size of the thrombus may

be evaluated and the area of the mass calculated. The ultrasound density of the mass may be classified as soft or dense. These echocardiographic parameters, in association with clinical data, may allow distinction between the pannus and thrombus.[14] This is essential to determine the underlying etiology of prosthetic dysfunction and to indicate optimal management of prosthetic valve obstruction (surgery versus thombolysis). Duration of symptoms, anticoagulation status, and ultrasound intensity of the mass obstructing mechanical prosthesis can in fact help to distinguish these two entities even though recognition of the pannus is still difficult. Patients with thrombus have shorter time intervals from valve insertion to malfunction, shorter duration of symptoms, and lower rate of anticoagulation. Pannus formation is more common in the aortic position. On echocardiography, thrombi are in general larger than the pannus and appear as a soft mass, whereas the pannus is more echo-dense. Moreover, TEE may also reveal other complications such as the coexistence of left atrial and/or left atrial appendage thrombi, which may be associated particularly with severe mitral prosthetic obstruction. Figure 17.4 shows an example of nonobstructive mitral prosthetic thrombosis.

Management of prosthetic valve thrombosis

Surgical intervention has been the traditional way to treat prosthetic valve thrombosis. More recently, thrombolysis has been reported to be an effective alternative treatment option.[14–18] Echocardiographic data (including Doppler data, disk mobility and thrombus/pannus) are extremely useful in indicating the best therapeutic intervention. Thrombus size is an independent predictor of outcome when thrombolysis is attempted.[19] Patients with small thrombus (area of the thrombus of <0.8 cm^2) have essentially no or minimal complications with thrombolysis. Patients with functional classes III and IV benefit even more by quantifying thrombus burden. In this subgroup with high surgical mortality, a small thrombus detected by TEE identifies patients with low complication and low death rates who may benefit from thrombolysis as first-line therapy. On the other hand, patients in functional classes III and IV with larger thrombi (>0.8 cm^2) need assessment of total surgical risk, but are, in general, candidates for valve surgery. Furthermore, disk motion may

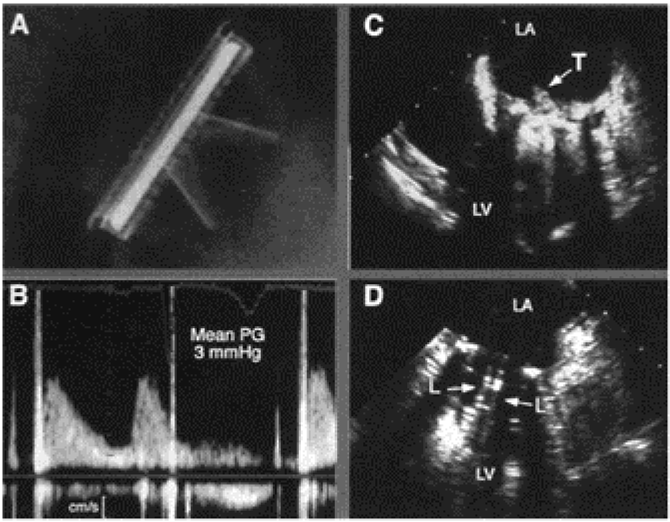

Figure 17.4 Patient with a bileaflet mitral valve prosthesis who had an acute cerebral ischemic attack. The two leaflets have normal opening angles (panel A, cinefluoroscopy; panel D, TEE). Despite normal mean transprosthetic gradient (panel B: mean gradient 3 mmHg), a large thrombus is clearly visualized by TEE (panel C).

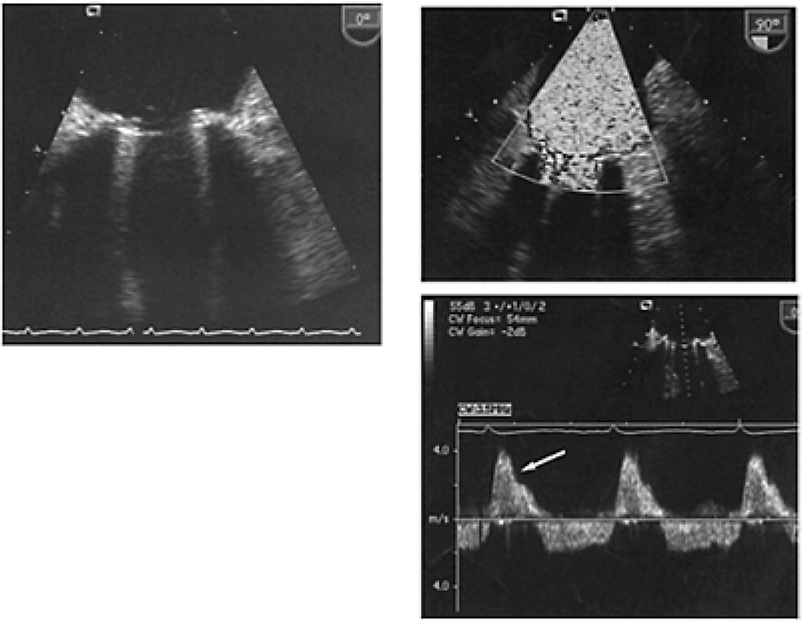

Figure 17.5 Embolization of the disk occluder of a convexoconcave Bjork-Shiley valve implanted in 1982. Left panel: transesophageal view of the mitral prosthesis. No occluder is identifiable in systole. Top panel right: color Doppler shows massive transprosthetic regurgitation. Bottom panel: transprosthetic continuous-wave Doppler, showing a saturated regurgitant signal in systole, a low maximal gradient (4 ms, corresponding to a maximal systolic ventriculoatrial pressure difference of 64 mmHg), and a late systolic notch or shoulder (arrow) due to massive rise of left atrial pressure during systole. (Reproduced from Novaro et al. J Am Sot Echocardiogr 2000; 13:417-30.[25] With permission from American Society of Echocardiography.)

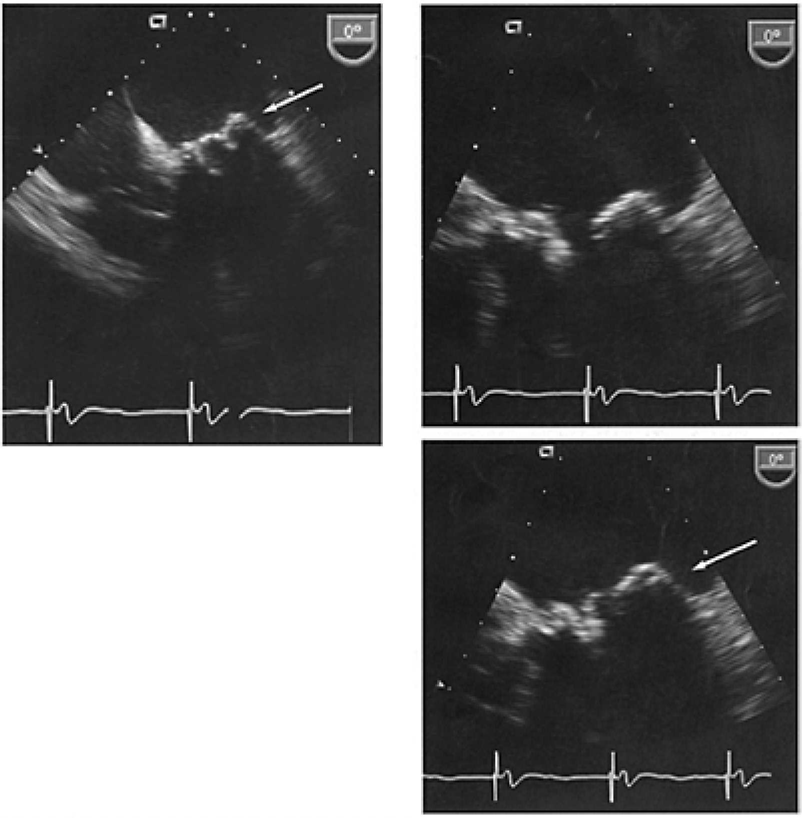

Figure 17.6 Dehiscence of a mitral bioprosthesis. Left panel: transesophageal four-chamber view in systole. An abnormal position of the bioprosthesis in systole is evident. Top right panel: open prosthesis in diastole; bottom right panel: closed prosthesis in systole. In the left and bottom right panel, the lateral circumference of the prosthesis is detached from the surrounding tissue (arrow). A comparison between the two right panels shows that the prosthesis is moving abnormally as a whole (‘rocking’). There was severe paraprosthetic regurgitation.

predict the efficacy of thrombolytic treatment.[20] In mitral valve thrombosis (bileaflet prostheses), hypomobile leaflets always recover regardless of symptom duration and extent of disk motion reduction, while, particularly in late prosthetic valve thrombosis, completely blocked leaflets do not respond to thrombolysis. Therefore, echocardiography (and fluoroscopy) may predict the results of thrombolysis by accurately defining the amount of restriction in disk motion. In bileaflet prostheses with one leaflet blocked and the other hypomobile (without contraindication due to very large thrombus), thrombolysis may be used to restore normal movement to the hypomobile leaflet to improve the patient's clinical and hemodynamic condition before operation.

These observations clearly indicate the importance of a complete transthoracic and transesophageal echocardiographic evaluation. The key to diagnosis and indication of medical or surgical treatment is not only the presence of an increased gradient, but also accurate evaluation of thrombus size, leaflet motion and coexistence of atrial thrombi.

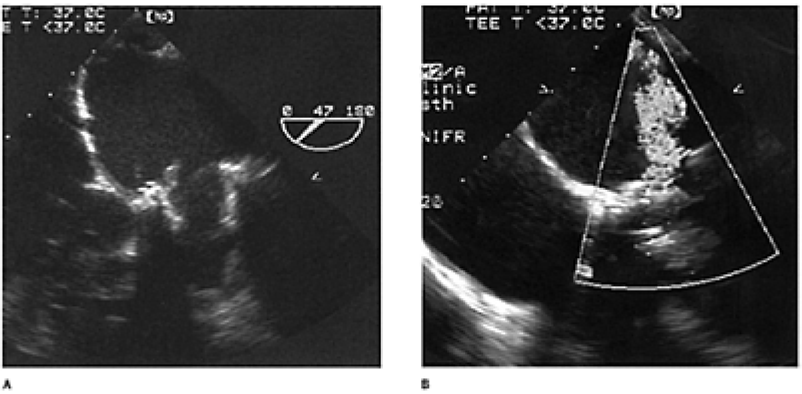

Figure 17.7 Severe transprosthetic regurgitation in a mitral bioprosthesis due to a torn leaflet (A). Note broad regurgitant jet and proximal convergence zone (B).

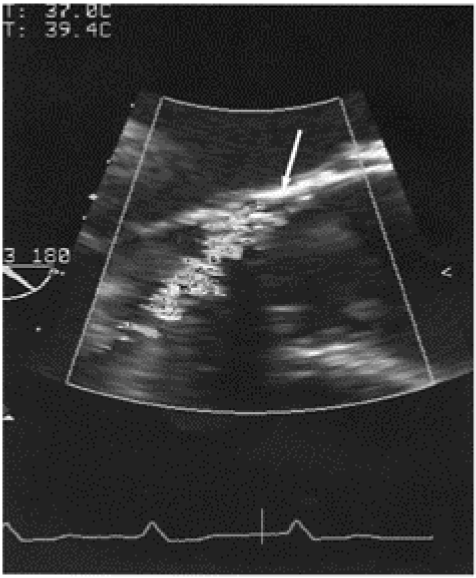

Figure 17.8 Moderate paravalvular leak in an aortic bioprosthesis. The paravalvular channel originating in the noncoronary sinus region is well visible (arrow).

Prosthetic regurgitation

All mechanical prostheses and practically all bioprostheses show minor regurgitation already when functioning normally. In mechanical prostheses, there is a closure backflow by the closing motion of the occluder (disk or leaflets), and an additional leakage flow after closure due to small leaks between the occluder and the ring or struts incorporated by design to ensure mobility and continuous flushing to prevent microthrombi. These small leaks may be picked up on spectral and color Doppler and show regurgitant jet patterns typical of valve types,[20] but they usually can be easily distinguished from major regurgitation. In bioprostheses, there is also minimal regurgitation similar to native 'physiologic' regurgitation. The following mechanisms are involved when severe and often acute regurgitation occurs in a valve prosthesis:

- Structural damage. The prototype of this mechanism is embolism of the occluder after strut fracture, leading to absence of any valve mechanism. For instance, this event occurred in some early Björk-Shiley valves (60° convexoconcave mitral valves manufactured in 1981-2; Figure 17.5), but it does occur rarely in other mechanical valves too. In bioprostheses, tears or ruptures in degenerated leaflets may also lead to sudden development of severe regurgitation (Figure 17.6).

- Ring dehiscence and large paravalvular leaks (Figures 17.7 and 17.8). Suture insufficiency may lead to abnor

Table 17.1 Echocardiographic signs of mitral and aortic prosthetic regurgitation (see also Chapter 13)

Echocardiographic signs of mitral prosthetic regurgitation

- Structural damage to the prosthesis, such as torn bioprosthetic leaflet, leaflet defect, or loss or immobilization of occluder in mechanical valves. The cause is degenerative, material failure (mechanical prostheses) or infective endocarditis.
- Paravalvular interruption of continuity at the level of the sewing ring. If a relatively small section of the ring circumference has lost its anchoring in the tissue, a paravalvular leak is present, with varying severity of regurgitation. If the section is big enough to produce rocking of the whole prosthesis, this is termed ‘dehiscence’ and severe regurgitation is always present.
- Forward flow is increased, with increased transmitral flow velocities, mimicking prosthetic obstruction. The prosthesis-specific pressure half-time, however, is not markedly changed. Note that regurgitation and true obstruction may coexist, as in prosthetic thrombosis.
- Proximal jet width is related to regurgitation severity as in native mitral regurgitation. A cutoff of 6–7 mm is usually chosen to separate severe, ‘surgical’ regurgitation from lesser degrees.
- The presence of a reproducible, large (>1 cm^2) proximal convergence zone (‘PISA’) indicates the presence of substantial regurgitation.
- Reversal of systolic pulmonary venous inflow (high specificity and modest sensitivity)
- In very severe regurgitation, there may be a ‘shoulder’ in the continuous wave signal of mitral regurgitation indicating a steep rise in left atrial pressure in late systole (Figure 17.1).

Echocardiographic signs of aortic prosthetic regurgitation

- Structural damage to the prosthesis, such as torn bioprosthetic leaflet, leaflet defect, or loss or immobilization of occluder in mechanical valves. The cause is degenerative, material failure (mechanical prostheses) or infective endocarditis.
- Paravalvular interruption of continuity at the level of the sewing ring, with consecutive paravalvular leak or dehiscence.
- Premature mitral valve closure (no A wave) and shortened mitral E wave deceleration.
- Forward flow is increased, with increased transaortic flow velocities, mimicking prosthetic obstruction. Note that regurgitation and true obstruction may coexist, as in prosthetic thrombosis.
- Proximal jet width is related to regurgitation severity, as in native aortic regurgitation, but usually is very difficult to gauge due to shadowing and jet artifacts.
- The presence of a reproducible, large (>1 cm^2) proximal convergence zone (‘PISA’) on the aortic side indicates the presence of substantial regurgitation.
- Shortened pressure half-time (<250 ms) of the continuous-wave Doppler signal of aortic regurgitation indicates severe regurgitation.
- Holodiastolic flow reversal in the ascending and particularly the descending aorta is a clear sign of severe aortic regurgitation (high specificity and moderate sensitivity).

mal motion of the entire prosthesis ('rocking'), which itself is mechanically intact, but produces regurgitation by a large gap between the periannular tissue and the prosthesis ring. There is a continuum from paravalvular leak to dehiscence, although the latter is usually restricted to valves that move abnormally in toto. Infective endocarditis (see below) of the prosthetic ring may lead to paravalvular leaks or dehiscence.

- Fixation of the occluder in a semiopen/semiclosed position by thrombosis, pannus, or other reasons, such as large vegetations. In these cases, there is often also an obstruction, but regurgitation may be clinically more evident.
- Infective endocarditis (see below) can directly destroy bioprosthetic leaflets, leading to prolapse, flail, defects, or rupture of entire bioprosthetic leaflets.

The typical echocardiographic signs of prosthetic regurgitation are summarized in Table 17.1. The clinical consequences of acute severe prosthetic regurgitation correspond to those of acute regurgitation of the respective native valve, with prominent backward failure and pulmonary congestion or edema for aortic and mitral prostheses and possible additional forward failure or full-fledged cardiogenic shock. In extremely severe regurgitation, typical murmurs such as the systolic murmur of mitral regurgitation may be absent or inaudible due to the massively reduced cardiac output.

Immediate echocardiography is mandatory. In most cases, the yield of TEE is markedly higher than transthoracic echo, and the clinical urgency makes a definitive diagnosis imperative. The techniques and methods to judge prosthetic regurgitation are not fundamentally different from the assessment of native valves[21] (see also Chapter 13; Table 17.1). However, artifacts and shadowing from valve prostheses and the fact that these patients very frequently have pathologically altered cardiac morphology make assessment considerably more difficult. It should be remembered that shadowing from a mitral prosthesis will often obscure mitral prosthetic regurgitation when viewed from an apical window. Use of parasternal and subcostal windows therefore is mandatory, and TEE is recommended. Contrary to its superiority for mitral regurgitation, assessment of aortic regurgitation may be very difficult by TEE; this is due, among other reasons, to difficult alignment of the continuous wave cursor with the direction of regurgitant flow, although often additional information is obtained (such as infective endocarditis, and abscess).

Prosthetic Infective endocarditis

Prosthetic valve endocarditis has an incidence of approximately 1% during the first year and less than 0.5% per patient and year thereafter. Due to the problems in imaging prosthetic valves, detection of vegetations and other signs of endocarditis is more difficult than in native valves. The suspicion of prosthetic valve endocarditis should therefore always prompt a TEE. Furthermore, the presence of a fever or systemic inflammatory signs in a patient with a prostheses should always raise the suspicion of prosthetic endocarditis. Rapid diagnosis is particularly critical if staphylococcal bacteremia is found. TEE allows thorough assessment, particularly of the atrial side of mitral prostheses, and also better evaluation of aortic prostheses. It has been convincingly

shown that the diagnostic accuracy of TEE for the detection of small vegetations, particularly abscesses, is markedly higher than that of transthoracic echo.[22,23] Typical signs of infective prosthetic endocarditis include the following:

- *Vegetations.* These are mobile, irregular masses attached to prosthetic structures, most often on the low pressure side (atrial side in mitral prostheses; ventricular side on aortic prostheses). Their maximal linear dimension correlates roughly with complications, particularly risk of embolism, and outcomes. Very large vegetations may cause obstruction, although this is rare. The echodensity correlates roughly with acuity of the disease, fresh vegetations having the echodensity of myocardium or less, and old vegetations often being calcified.
- *Abscesses.* These are perivalvular cavities or localized perivalvular tissue thickening. Their presence and extent are greatly underestimated by transthoracic echo and often even by TEE. Ring abscesses are typical of mechanical prostheses. The cavities may or may not have access to heart chambers or the ascending aorta and may create fistulas between heart chambers.
- *Defects (holes) in bioprosthetic leaflets creating regurgitation.*
- *Paraprosthetic leaks and prosthetic dehiscence.* This has been discussed in the section on 'Prosthetic regurgitation'.
- *Pericardial effusion.* Small effusions often signal bacterial tissue invasion.

Since prosthetic endocarditis practically mandates urgent surgical valve replacement due to its poor responsiveness to antibiotic treatment, it is very important to furnish to the surgeon complete information about the extent of endocarditis and the presence of complications. All valves have to be systematically evaluated for the signs of endocarditis.

On the other hand, a negative TEE is a strong argument against the presence of infective endocarditis, but it does not completely rule out the disease. There may be vegetations too small to be visualized, acoustic shadowing, or artifacts from the prosthesis. However, a negative repeat TEE after a few days (5–7 days) has a very high negative predictive value to rule out endocarditis.[24]

References

1. Vongpatanasin W, Hillis D, Lange R. Prosthetic heart valves. N Engl J Med 1996; 335:407–16.
2. Kotler MN, Mintz GS, Panidis I, et al. Noninvasive evaluation of normal and abnormal prosthetic valve function. J Am Coll Cardiol 1983; 2:151–73.
3. Jones M, McMillan ST, Eidibo EE, Woo YR, Yoganathan AP. Evaluation of prosthetic heart valves by Doppler flow imaging. Echocardiography 1986; 3:513–25.
4. Nellessen U, Schittger I, Appleton CP, et al. Transesophageal two-dimensional echocardiography and color Doppler flow velocity mapping in the evaluation of cardiac valve prostheses. Circulation 1988; 78:848–55.
5. Nanda NC, Cooper JW, Mahan EF,Fan P. Echocardiographic assessment of prosthetic valves. Circulation 1991; 84 (Suppl I): I-228–39.
6. Khanderia B. Transesophageal echocardiography in the evaluation of prosthetic valves. Am J Cardiol Imaging 1995; 9:106–14.

7. Montorsi P, De Bernardi F, Muratori M, Cavoretto D, Pepi M. Role of cine-fluoroscopy, transthoracic and transesophageal echocardiography in patients with suspected prosthetic heart valve thrombosis. Am J Cardiol 2000; 85:58–64.
8. Montorsi P, Cavoretto D, Parolari A, Muratori M, Alimento M, Pepi M. Diagnosing prosthetic mitral valve thrombosis and the effect of the type of prostheses. Am J Cardiol 2002; 90:73–6.
9. Baumgartner H, Khan S, DeRobertis M, Czer L, Maurer G. Discrepancies between Doppler and catheter gradients in aortic prosthetic valves in vitro: a manifestation of localized gradients and pressure recovery. Circulation 1990; 82:1467–75.
10. Baumgartner H, Schima H, Tulzer G, Kuhn P. Effect of stenosis geometry on the Doppler-catheter gradient relation in vitro: a manifestation of pressure recovery. J Am Coll Cardiol 1993; 21:1018–25.
11. Flachskampf FA, Hoffmann R, Franke A, et al. Does multiplane transesophageal echocardiography improve the assessment of prosthetic valve regurgitation? J Am Soc Echocardiogr 1995; 8:70–78.
12. Daniel W, Mugge A, Grote J, et al. Comparison of transthoracic and transesophageal echocardiography for the detection of abnormalities of prosthetic and bioprosthetic valves in the mitral and aortic position Am J Cardiol 1993; 71:210–15.
13. Barbetseas J, Nagueh SF, Pitsavos C, et al. Differentiating thrombus from pannus formation in obstructed mechanical prosthetic valves: an evaluation of clinical, transthoracic and transesophageal echocardiographic parameters. J Am Coll Cardiol. 1998; 32:1410–17.
14. Roudaut R, Labbe T, Lorient-Roudaut M, et al. Mechanical cardiac valve thrombosis: is fibrinolysis justified? Circulation 1992; 86 (Suppl II):8–15.
15. Lengyel M, Fuster V, Keltai M, et al. Guidelines for management of left-sided prosthetic valves thrombosis: a role for thrombolytic therapy. J Am Coll Cardiol 1997; 30:1521–6.
16. Lengyel M, Vandor L: The role of thrombolysis in management of left-side prosthetic valve thrombosis: a study of 85 cases diagnosed by transesophageal echocardiography. J Heart Valve Dis 2001; 10:636–49.
17. Shapira Y, Herz I, Vaturi M, Porter A, Adler Y, et al. Thrombolysis is an effective and safe therapy in stuck bileaflet mitral valves in the absence of high-risk thrombi. J Am Coll Cardiol 2000; 35:1874–80.
18. Tong A, Roudaut R, Ozkan M, et al. Transesophageal echocardiography improves risk assessment of thrombolysis of prosthetic valve thrombosis: results of the international PRO-TEE registry. J Am Coll Cardiol 2004; 43:77–84.
19. Montorsi P, Cavoretto D, Alimento M, Muratori M, Pepi M. Prosthetic mitral valve thrombosis: can fluoroscopy predict the efficacy of thrombolytic treatment? Circulation 2003; 108 (Suppl II): 11–79–84.
20. Flachskampf FA, Guerrero JL, O'Shea JP, Weyman AE, Thomas JD. Patterns of normal transvalvular regurgitation in mechanical valve prostheses. J Am Coll Cardiol 1991; 18:1493–8.
21. Zoghbi WA, Enriquez-Sarano M, Foster E, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. J Am Soc Echocardiogr 2003; 16:777–802.
22. Daniel WG, Mügge A, Martin RP, et al. Improvement in the diagnosis of abscesses associated with endocarditis by transesophageal echocardiography. N Engl J Med 1990; 324:795–800.
23. Daniel WG, Mügge A, Grote J, et al. Comparison of transthoracic and transesophageal echocardiography for detection of abnormalities of prosthetic and bioprosthetic valves in the mitral and aortic positions. Am J cardiol 1993; 71:210–15.
24. Sochowski RA, Chan KL. Implication of negative results on a monoplane transesophageal echocardiographic study in patients with suspected infective endocarditis. J Am Coll Cardiol 1993; 21:216–21.
25. Novaro GM, Robbins MA, Firstenberg MS, Prior DL, Stewart WJ, Rodriguez LL. Disk embolization of a Bjork-Shiley convexo-concave mitral valve: a cause of sudden cardiovascular collapse and mesenteric ischemia. J Am Soc Echocardiogr 2000; 13:417–20.

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The main heading ‘echocardiography’ includes all subtypes.